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VOL. 32, NO. 1 JULY, 1959

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20, 21, 22, 23 September 1959

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AMERICAN COLLEGE
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1. Rand, N. T. and Collins, V. K.: Food Technology *12*:585 (Nov.) 1958.

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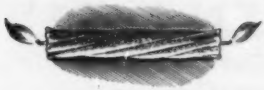

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1. Case reports on file, Wyeth Laboratories. 2. Parks, R.V., and Moessner, G.F.: Dual Approach to Patient Care, Scientific Exhibit, A.A.G.P., April, 1959.

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(FORMERLY THE REVIEW OF GASTROENTEROLOGY)

The Pioneer Journal of Gastroenterology, Proctology and Allied Subjects in the United States and Canada

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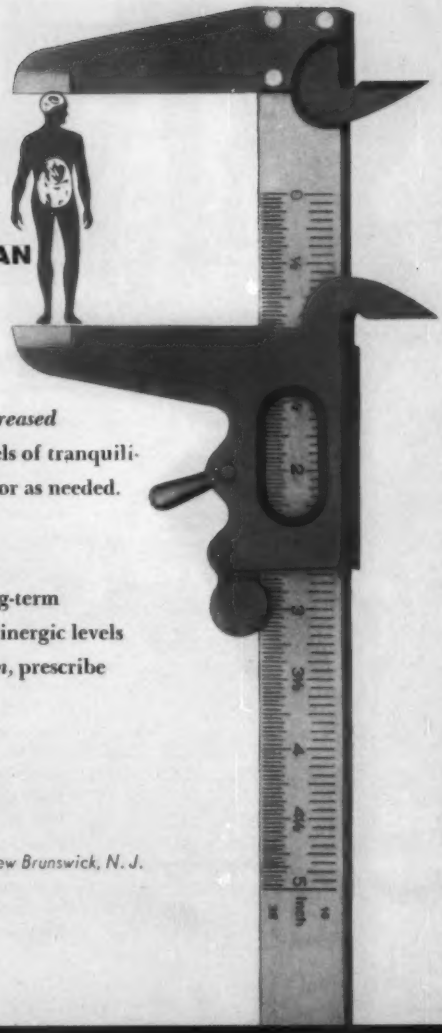

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Pylorospasm or cardiospasm	11	2
Irritable bowel	11	—
Biliary tract dysfunctions	11	1
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Fair	56	—
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References: 1. Winkelstein, A.: Am. J. Gastroenterol., in press. 2. Leming, B. H., *Jr.*: Clin. Med. 6:423 (March) 1959. 3. McHardy, G., et al.: Paper presented at Postgraduate Course in Gastroenterology, University of California School of Medicine, San Francisco, Calif., January 27, 1958. 4. Strub, I. H., and Carballo, A.: To be published. 5. Data in Roerig Medical Department files. 6. Steigmann, F.: To be published. 7. Schuller, E.: Gaz. des Hôpitaux *10*:391 (Apr. 10) 1957. 8. Farah, L.: Internat. Rec. Med. *169*:379 (June) 1956. 9. Harrisson, J. W. E., et al.: Paper presented at the 4th Pan-American Congress of Pharmacy and Biochemistry, Washington, D. C., November 3-9, 1957.

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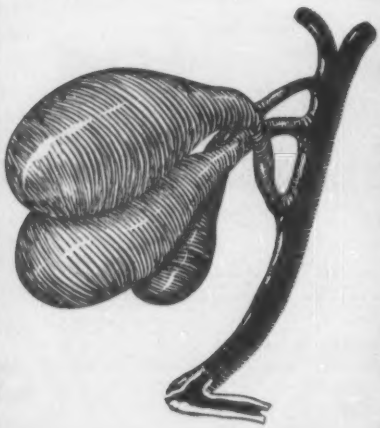

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(1) Beckman, H.: Drugs: Their Nature, Action and Use, Philadelphia, W. B. Saunders Company, 1958, p. 425.
(2) Biliary Tract Diseases, M. Times *85*:1081, 1957.

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THE American Journal OF Gastroenterology

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SURGERY OF BENIGN LESIONS OF THE ESOPHAGUS*

LAWRENCE H. STRUG, M.D.
WILLIAM LEON, M.D.
and
BERT A. GLASS, M.D.
New Orleans, La.

In the past two decades diseases of the esophagus have assumed a prominent position in the surgical mind. Lesions which heretofore had either been considered extremely rare, overlooked, or hopeless have become important in the eyes of every physician. Undoubtedly this new viewpoint was brought about by an increased awareness of such entities, by improvement in diagnostic technics and by the vast improvement in anesthesia, which allowed a more radical surgical approach to these problems. The addition of chemotherapeutic agents has helped considerably in this regard.

Pathologic conditions of the esophagus may involve the esophagus at any age. These range over a wide field from the congenital anomalies to the acquired states which are seen in middle and older age groups. It has become obvious in recent years that the esophagus is extremely important in consideration of differential diagnosis of disease states of the thorax and upper abdomen. The stimulation given to the problems of the esophagus can in large measure be attributed to the work of Sweet[1], Torek[2], Haight[3] and others[4,5,6].

CONGENITAL ANOMALIES

Congenital malformations of the esophagus are not uncommon. These may be limited to the esophagus itself or because of its embryological origin may involve the trachea. In many instances the anomalies are multiple, involving the heart and gastrointestinal tract. When this occurs, the situation is usually hopeless and survival is rarely beyond a few days. The advances made in thoracic

*Presented before the Course in Postgraduate Gastroenterology of the American College of Gastroenterology, New Orleans, La., 23, 24, 25 October 1958.

Department of Surgery, Louisiana State University School of Medicine, New Orleans, La.

surgery in the past generation have certainly altered the outlook on some of these conditions, particularly congenital atresia of the esophagus.

The types involving the esophagus alone are[1]:

1. Total absence of the esophagus (extremely rare)
2. Partial absence of the esophagus
3. Complete absence of the esophageal lumen
4. Partial absence of the lumen
5. Double esophagus (reduplication)
6. Congenital webs and stenosis. This type is the most frequently encountered malformation of the esophagus alone. Extremely few of this type are compatible with survival. If the web or stenosis is not complete so that the infant survives long enough for it to be recognized, surgical intervention may result in survival.

Tracheoesophageal Fistulas

Malformations involving the trachea are more common. The most frequent of this type is atresia of the esophagus with tracheoesophageal fistula. This group comprises 70 to 80 per cent of all anomalies of the esophagus.

Five main types may be present, although there may be a number of variations:

Type I:—The upper portion of the esophagus ends as a blind pouch and the lower esophageal segment begins as a blind pouch, usually 3-4 vertebral segments lower (Fig. 1A).

Type II:—The upper esophageal segment ends in a fistulous tract which enters the trachea immediately above the bifurcation, and the lower esophageal segment resembles Type I (Fig. 1B).

Type III:—The upper segment ends in a blind pouch as in Type I, and the lower segment communicates with the trachea immediately above its bifurcation (Fig. 1C).

Type IV:—is similar to Type III except that the fistulous tract is located at the carina (Fig. 1D).

Type V:—The upper and lower segments both communicate with the trachea (Fig. 1E). There are a number of variations of this type.

Types III and IV are the most common. Esophageal atresia with tracheoesophageal fistula is not as uncommon as was formerly believed. It is estimated

by some that the frequency is approximately one in every 2,500 births. To Cameron Haight[3] must go the credit for the first successful operation of the direct type, which encompassed division of the fistulous tract and end-to-end anastomosis of the esophagus. The approach initially was extrapleural, but the present approach is directly through the right hemithorax.

The clinical manifestations of this anomaly are such that if one has an acute awareness, the diagnosis can be made early, i.e. a few hours after birth. The most important objective finding is unusual salivation. Choking overfeedings with coughing paroxysms and cyanosis, should immediately suggest the possibility of an atresia of the esophagus with tracheoesophageal fistula. Steps should then be immediately taken to substantiate the diagnosis, which consist of instillation of an opaque material other than barium into the proximal end of the esophagus. Absence of air in the gastrointestinal tract should make one suspicious that the anomaly may be an atresia or absence of the esophagus without tracheoesophageal fistula.

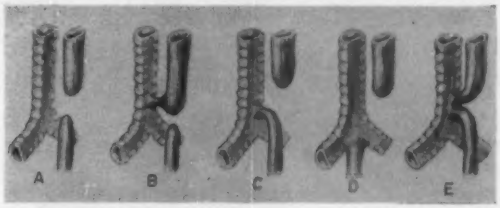

Fig. 1—Five main types of tracheoesophageal fistula. Type C is the most common.

It must be stressed that the preoperative preparation of the patient and the postoperative nursing care are of the most vital importance, as they directly affect the morbidity and mortality.

On rare occasions when on exploration, an anastomosis in continuity cannot be made, the fistula should be divided, the thorax closed, and gastrostomy and cervical esophagostomy performed. When the infant reaches an older age, some form of substitution procedure can be instituted, as it is obvious that the infant could not withstand such an extensive surgical procedure immediately.

Congenital Cysts and Duplications

As a result of the pinching-off of the esophagus from the trachea in early embryonic life, there may develop a duplication in the wall of the esophagus or alongside it. These may be isolated, without any communication into the major tube or may have communications at both ends. The esophageal musculature may be a continuous layer which covers both the normal esophagus and the duplication, without any line of cleavage. The separation may on the other hand be complete, so that each is covered with separate musculature.

Most duplications present symptoms during the first two years of life. Some patients are asymptomatic, and the only manifestation may be a cyst-like structure present in the posterior mediastinum which is adjacent to the esophagus and is recognized on routine roentgen examination.

Such symptoms as regurgitation, cough, respiratory embarrassment and dysphagia may be present. Occasionally those cysts that communicate with the esophagus may become infected, increasing the symptomatology. This entity must be differentiated from dermoids, teratomas, bronchogenic and pericardial cysts, which as a rule are located in the anterior mediastinum.

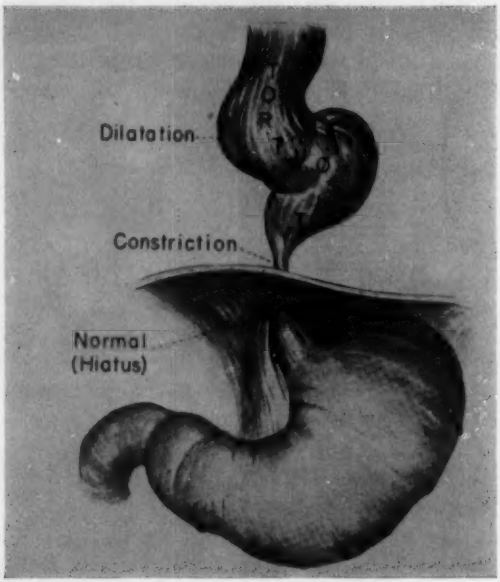

Fig. 2—Megaesophagus, saccular type showing marked dilatation and tortuosity of the esophageal segment.

The treatment of choice, when recognized, is complete surgical excision. This may be difficult as the duplication is often intimately connected to the esophagus when a common muscular wall is present. One of the alternatives is to make a large internal opening into the esophagus or marsupialize the cyst as suggested by Gross[7].

Megaesophagus (Achalasia)

This term is applied to a peculiar condition of the esophagus, the etiology of which is not known. It is characterized by a marked degree of dilatation and

hypertrophy of the major part of the esophagus above a constricted distal segment. Various names have been accorded this condition, such as, "idiopathic dilatation", "congenital dilatation", "cardiospasm", "phrenocardiospasm", "achalasia", and others. It is apparent that none of these terms are satisfactory. In 1915 Hurst proposed the term achalasia and to many, it is more accurately descriptive of the abnormal physiological behavior of the esophagus in this disease. Many theories have been advanced as to its cause, but none have been proven. It may exist at any age, and no one can judge how long this condition existed before recognition.

Two clinical types can be recognized. These can be distinguished on the basis of the clinical history, x-ray examination and the anatomical findings at operation. These are the sigmoid type of dilatation and the fusiform (Fig. 2).

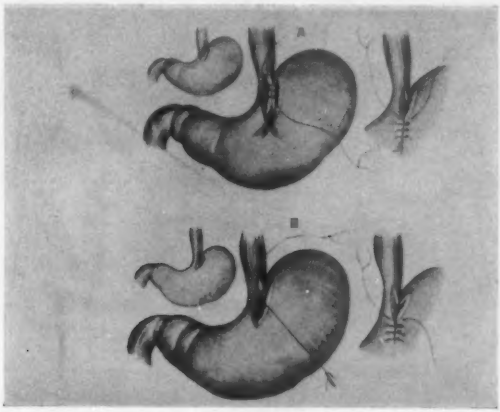

Fig. 3—Diagramatic sketch of the Heyvrowsky (A) and Grondahl (B) procedures occasionally performed in achalasia of the esophagus.

The majority are of the sigmoid type (70 per cent)[8]. The esophagus often becomes redundant, tremendously dilated and bulges to the right. In contrast the lower segment is unusually small, appearing like a child's esophagus, and the muscular layer is characteristically thin.

It is impossible in most instances to determine the onset of the symptoms. The swallowing function may have been slightly impaired for some time. Occasionally, even with a markedly dilated esophagus, the symptoms may be nonexistent or minimal. In the majority, however, dysphagia becomes progressive, and often associated with a dull deep substernal discomfort. As time goes on the discomfort becomes more pronounced and it is complicated by regurgitation. As the esophagus enlarges the regurgitation may decrease, because food packs into the dilated esophagus. Occasionally severe malnutrition may occur.

In order to get a satisfactory roentgen examination, the esophagus must be emptied. In the severe cases there is little evidence of peristalsis. Esophagoscopy should be performed on all cases. It is often difficult if not impossible to pass the esophagoscope through the cardia. In long-standing achalasia, leukoplakic areas on the mucosal surface may be evident. The differential diagnosis usually does not present any serious problem. The chief diagnostic differentiation is that of cancer. It cannot be made except by roentgen examination and esophagoscopy. On occasions it may be necessary to explore the esophagus in order to differentiate between carcinoma and achalasia. Spasm of the lower esophageal segment must be differentiated from achalasia. As a rule it does not yield the good results as noted in achalasia by dilatation.

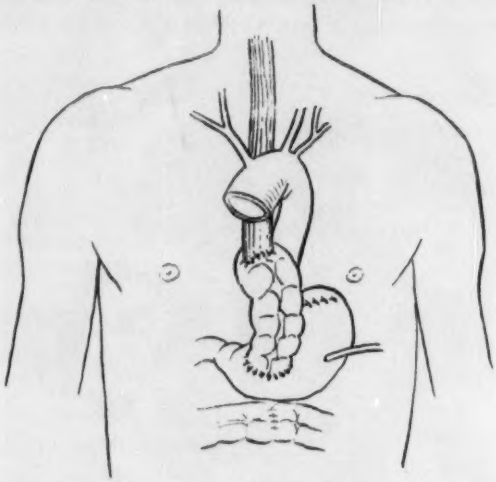

Fig. 4—Illustration of colon substitution for an esophageal segment employing the mid-transverse colon.

Treatment is divided into medical and surgical phases. Initially, in other than the far advanced cases, medical therapy is the procedure of choice. Dietary measures may be helpful. Medication aimed at relaxing the distal esophageal segment is of little help. The best form of treatment for the majority of patients is the methodical passage of a bougie through the cardia. This, coupled with various types of dilators such as the mercury and hydrostatic types, has been extremely helpful. The treatments at first are carried out every few days and then the interval is gradually lengthened. According to Allison 70 per cent can be cured and an additional 20 per cent relieved[9].

Many operations based on theoretical considerations have been tried and found undesirable. A number of procedures directed at altering the anatomy of the lower constricted esophageal segment have been tried and given up. Among

these are the group classed under so-called cardioplastics. These include the Heyvrowsky and the Grondahl procedures[8] (Fig. 3). The complicating esophagitis, ulceration and stenosis which developed, led to abandonment of these procedures by most individuals.

In 1914 Heller first described the procedure of esophagomyotomy. This has enjoyed some degree of success. In a review of these cases at the Mayo Clinic the Heller procedure offered the greatest benefit to a fair number of patients[8]. This may be performed through the abdomen or left thorax. In an occasional case which has not responded to conservative surgical measures, esophagogastric resection may be mandatory.

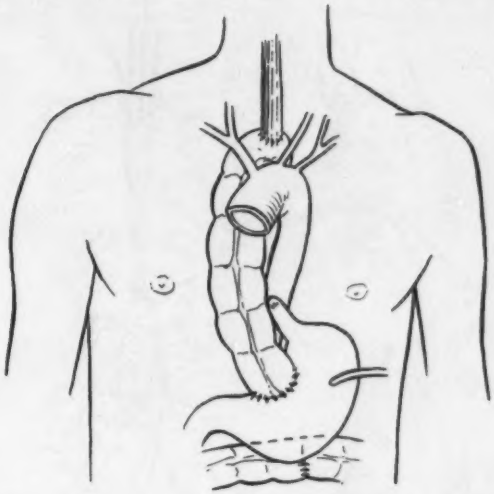

Fig. 5—Colon substitution replacing entire thoracic esophagus, employing transverse colon.

Complications

It should be stressed that in patients with long-standing achalasia, complications are fairly common. The most frequent is that of repeated episodes of pneumonitis which are due to aspiration. This is occasionally complicated by bronchiectasis and lung abscess. It is extremely interesting that there is an increasing number of cases of this type with specific pulmonary infections. In our experience we have had two such patients. One case had an unresolved pneumonitis which upon resection proved to be nocardiosis, and the second one actinomycosis. Other complications that may develop are hemorrhage from an ulcerated area, acute perforation and mediastinitis.

When less formidable surgical procedures have been unsuccessful one should resort to total esophagectomy with colon substitution. Wangensteen has

advised partial esophageal resection including the constricted segment, removal of the acid-bearing portion of the stomach and pyloromyotomy[10].

Diverticula

Diverticula of the esophagus are divided into two types, pulsion and traction. They occur mainly in the cervical esophagus and in the area of the esophagus distal to the bifurcation of the trachea. The most common type is that which occurs in the pharyngoesophageal area. This in effect is a true diverticulum, in that the mucous membrane pouches out in a defect in the musculature. It be-

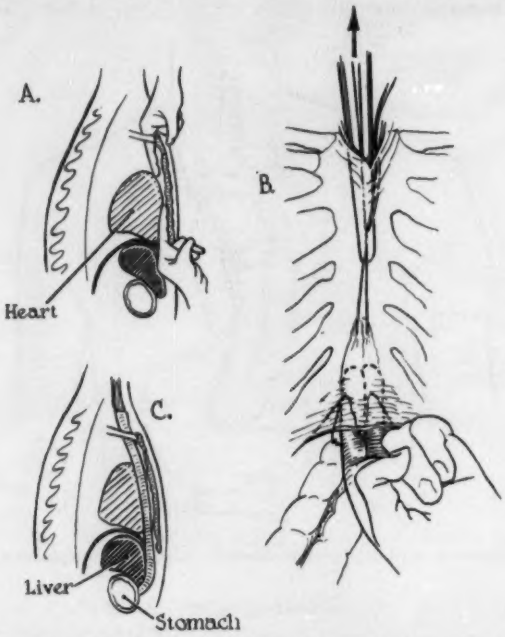

Fig. 6—Method illustrating retrosternal tunnel for colon substitution.

comes elongated and descends along the esophagus. The size of the orifice to the diverticula varies considerably. In the early development there is no real characteristic symptomatology. As the size of the diverticulum progresses the symptoms become more apparent. Some of the most prominent are intermittent dysphagia, feeling of fullness in the throat, and actual obstructive symptoms. There usually develops a fullness in the neck on the side of the pouch, when the diverticula assume considerable size.

On roentgen examination it has the appearance of a pouch suspended from the esophagus. It may or may not have a fluid level. Physical examination of the

individual does not as a rule contribute anything toward establishing a diagnosis unless the pouch is of immense size. The diagnosis is usually made by roentgen examination and esophagoscopy. It is not unusual in individuals with large diverticula of the pharyngoesophageal type to have repeated episodes of aspiration pneumonitis.

Traction diverticula usually occur in the mid-esophagus at the level of the bifurcation of the trachea. They are caused by adhesions of the esophageal wall and the juxtapositioned inflamed lymph nodes. As contraction occurs, traction is exerted on the wall of the esophagus, causing a small tent-like out-pouching. Routine esophagograms have shown that the incidence is higher than heretofore expected.

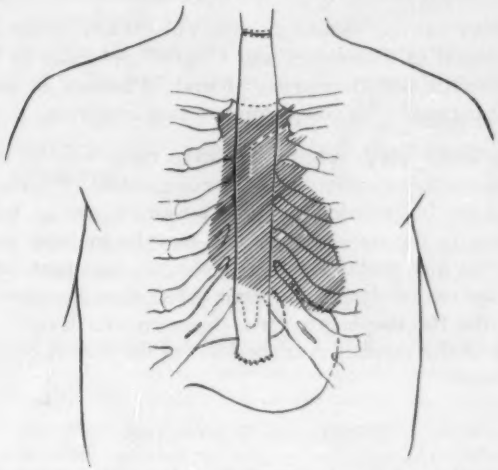

Fig. 7—Position of right half of the colon retrosternally, in colon substitution of the entire esophagus.

They rarely produce symptoms, as they are usually small and the traction is upward or horizontal. Surgery as a rule is unnecessary. Occasionally they may be associated with carcinoma of the esophagus, therefore this condition must be ruled out by esophagoscopy.

Epiphrenic diverticula are not uncommon. They may develop in any portion of the lower esophageal segment. They have been classified as of the pulsion type and have been frequently described in association with hiatal hernia.

The diverticulum readily becomes dependent and therefore is prone to cause symptoms such as substernal pain, dysphagia and regurgitation. It is often complicated by ulceration, perforation and hemorrhage.

Treatment

All pharyngoesophageal diverticula will eventually require surgical treatment. Up to rather recently the two-stage repair was the procedure of choice. Within recent years, it has been shown that a one-stage repair can be satisfactory in most instances. As mentioned previously traction diverticula as a rule do not require surgical excision.

The smaller epiphrenic diverticula rarely require any surgical treatment. If the pouch is large or is causing symptoms, the treatment is surgical. Primary excision is the treatment of choice. The approach is much better through the left thorax.

Benign Tumors of the Esophagus

Benign tumors of the esophagus are rare. Their relative frequency is attested by the report of Calmenson and Clagett[11], 44 cases in 7,459 autopsies. In the living, Moersch and Harrington[12] found 59 tumors as the cause of dysphagia in 18,459 patients who complained of that symptom.

The division into types is arbitrary. Various types may be observed including cysts, leiomyomata, neurofibromata, hemangiomata, fibromata, papillomata and mucosal polyps. In recorded series leiomyomata are by far the most frequent. These arise in the muscle layer and may be multiple or conglomerate. They may occur in any portion of the esophagus, but most frequently in the lower third. Symptoms of dysphagia occur when they become very large and almost circumscribe the esophagus (arise from circular layer). Esophagoscopy shows narrowing of the esophagus at the level of the tumor. No mucosal ulceration occurs, however.

Esophageal Substitution

The use of portions of the alimentary tract for replacement of the esophagus is becoming more widespread. From a historical viewpoint, esophageal substitution goes back many years. Nearly half a century has elapsed since Torek[2] reported the first successful resection of the thoracic esophagus. At that time external tubes were used to replace the esophagus. This was fraught with many complications. Many additional methods were utilized since that time, and did not prove entirely satisfactory. This was particularly true in resection for benign disease where a long life expectancy was anticipated. The work of Phemister and Adams[5], using the stomach as an esophageal replacement, first attempted in 1938 for carcinoma of the fundus of the stomach, has been widely used both in palliative resection for carcinoma and benign disease. It has, however, numerous disadvantages.

Sweet[1] and Garlock[6], employed the stomach in higher and higher transplantations. The stomach has distinct advantages, it is readily available, has a

good blood supply and can be readily mobilized. The development of peptic esophagitis, however, regurgitation of gastric contents into the pharynx in high resections, and the generally unhappy state of many of these individuals in regard to their eating habits are distinct disadvantages. Experimental work has lent support to the clinical experience that the stomach is not a good substitute for the esophagus[10].

A subcutaneous jejunal transplant was first used successfully by Roux in 1907[13]. After a lapse of a number of years the work of Rienhoff[13], Harrison[14], and Robertson[15] lent support to Roux's idea. The latter group placed the jejunum intrathoracically. The short vascular arcades made it extremely difficult to obtain a long healthy segment. This usually resulted in gangrene of the proximal portion of the jejunum with resultant leaks at the proximal anastomotic site. If this was in the neck, the resultant infection could easily be controlled by incision and drainage. This always necessitated a secondary anastomotic procedure. If, however, it occurred in the mediastinum, the consequences could prove fatal.

The use of inert plastic tubes as an esophageal bridge as advocated by Berman[16], has proved disastrous in our hands both experimentally and clinically and was readily abandoned.

The use of the colon as a replacement for the esophagus is not new. Kelling in 1911[17] reported a successful case of colon transplant for esophageal disease. Numerous reports followed particularly in Europe in which the colon was used successfully as a subcutaneous tube.

Replacement of the esophagus by colon within the thorax was first reported by Rudler in 1951[18]. He did this in a case of carcinoma of the esophagus placing the colon retrosternally. The distal end of the colon was anastomosed to the jejunum. In this country in 1952, Robertson[15] reported a successful case of esophageal substitution between the esophagus and jejunum. Other numerous reports followed[14,15]. Additional patients with carcinoma of the esophagus so treated were reported by Mahoney and Sherman in 1954[19].

The criteria of excellent esophageal substitution as laid down by Neville[20] are as follows: "It should have sufficient length, be assured of continuing viability, and the lining be resistant to acid peptic digestion."

It appears that the colon meets these conditions best and in addition offers other distinct advantages. It has an excellent blood supply, and the marginal arteries are such that extremely long segments can be mobilized and suspended on a pedicle of the middle colic artery.

Replacement of the esophagus by segments of colon, especially for benign lesions offers a good measure of success for several reasons. Experimentally it has been demonstrated that the colon is more resistant to acid gastric secretion than the jejunum or esophagus. Substitution can then be accomplished without

by-passing the stomach (Figs. 4 and 5). Sufficient length of colon can be mobilized and viability determined prior to section of the colon. Furthermore, adequate preparation with antibiotics is helpful.

We have used esophageal substitution in 13 cases for benign disease, and would herein report our experiences. In three cases jejunal transplants were used. In the remainder, colon substitutions were employed. Up to the present time, we have not used it with any enthusiasm in carcinoma of the esophagus. The definite advantage in the use of colon substitution for benign disease is that it is often not necessary to perform an intrathoracic resection of the esophagus. This shortens the procedure considerably and directly affects the morbidity and mortality attendant with prolonged surgery.

On our thoracic service, esophageal substitution has been employed principally for benign disease. Although our series has not reached the proportion of others reported, it has been sufficient in number to allow us to formulate certain ideas concerning this procedure.

Either the colon or jejunum should be used for intractable benign disease such as stricture of the esophagus which has not responded to conservative measures. The possibility of substitution of the colon for the esophagus in cases of achalasia which have failed to respond to more conservative surgery is another problem to which we have given serious thought. We have such a case at present on our service. Here it would be necessary to excise the esophagus. In the cases of extensive strictures, however, the esophagus can be left *in situ*. The colon is brought up retrosternally into the neck (Fig. 4), the esophagus divided in the neck and the distal end closed with two layers of sutures. The proximal end of the colon is then anastomosed to the proximal end of the esophagus (Fig. 7). The necessity of opening the thorax is thus obviated.

Two cases of colon substitution in intractable esophageal strictures are abstracted from our records and herein reported.

Case Reports

Case 1:—G. C., 38 years, colored male, admitted to the Louisiana State University EENT Division of Charity Hospital, New Orleans, La. on 21 April 1958 with a history of accidental ingestion of a glass of lye five weeks prior to admission. He was immediately treated by a local physician, who had him hospitalized. His stomach was washed and a weak solution of vinegar was instilled. He remained in the hospital for ten days. Five days following his discharge from the hospital he noticed progressive increasing difficulty in swallowing solids, then liquids. He vomited his meal usually four hours following its ingestion. In the three weeks prior to admission he lost 38 pounds. Esophagoscopy on 23 April 1958 revealed burns of the pharynx and esophagus. A stricture was present just below the level of the cricopharyngeus. Lipiodol study of the

esophagus revealed narrowing of the entire esophagus, more pronounced just below the cricopharyngeus and the distal third. Because of the continued nausea and vomiting a gastrointestinal x-ray series was performed which revealed in addition to the strictures a complete pyloric obstruction.

In view of this finding, it was decided to forego conservative management on this patient. Accordingly he was transferred to the thoracic service on 30 April 1958. Following preparation by gastric decompression through a small nasal tube inserted into the stomach, supportive therapy and preparation of the colon, he was operated upon on 5 May 1958.

Abdominal exploration revealed a complete pyloric obstruction. The right half of the colon was completely mobilized, divided in the mid-transverse colon and the terminal ileum, approximately four inches from the ileocecal valve. The right colon was brought behind the stomach, and retrosternally into the neck. The cervical portion of the esophagus was divided above the stricture area and the distal end closed. The colon was then anastomosed to the hypopharynx. The distal end of the colon was anastomosed to the anterior wall of the stomach. A gastroenterostomy was then performed because of the pyloric obstruction. The operative procedure was completed with a gastrostomy and ileotransversostomy.

Convalescence was uneventful except for a minimal right pneumothorax. He was discharged from the hospital on the sixteenth postoperative day, eating a full diet. Follow-up in December 1958 revealed that he is slowly gaining weight and is feeling fine.

Case 2:—C. F., 3 years, colored female, ingested a solution of lye on 15 May 1958 and was hospitalized for one week at the Monroe, Louisiana Charity Hospital. Convalescence was satisfactory until June 1958 when she began having dysphagia. She was again hospitalized and the suspected stricture of the esophagus was confirmed by a barium swallow. Gastrostomy was performed. She was then referred to the New Orleans Charity Hospital and was admitted to the Louisiana State University EENT Division. Repeat esophagram revealed a stricture starting at the level of T_4 and extending downward to the esophagogastric junction.

Attempts at repeated dilatation were only moderately successful. Esophagoscopy revealed that the stricture started at 14 cm. from the incisor teeth. In view of the extreme difficulty in the management of this child with dilatation and the large extent of the stricture, surgery was consulted, and a decision made in favor of an attempt at colon substitution of the strictured esophagus.

She was transferred to the thoracic service, and after proper colon preparation, the child was submitted to surgery on 24 November 1958, at which time the right half of the colon was brought retrosternally and anastomosed to the cervical esophagus in the usual manner.

Summary

Benign diseases of the esophagus including congenital anomalies, cysts and duplications, achalasia, diverticula, benign tumors of the esophagus and colon substitution for certain benign lesions have been reviewed. Emphasis on surgical management of a number of these disease states has been stressed.

Esophageal substitution in 13 cases of benign stricture of the esophagus was performed. The jejunum was used in three cases and the colon in ten.

Two cases illustrating colon substitution are reported.

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THE SURGICAL APPROACH TO HIATAL HERNIA IN THE PATIENT PAST 60 YEARS OF AGE*

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Hiatal hernia increases in frequency with each decade in life. The hernia also becomes larger with the passage of time and the esophageal changes become more marked.

In spite of accumulating knowledge, confusion still exists concerning surgical indications and results in the geriatric patient.

This study was undertaken to evaluate the experience in the elderly patient with esophageal hiatal hernia at Charity Hospital, New Orleans, La. from 1943 through 1955.

Surgical Considerations

Classifications of hiatal hernia assume importance because the reflux of gastric contents into the lower reaches of the esophagus varies with the type of hernia. Akerlund[1] originally classified hiatal hernia into: 1. esophagogastric type, in which the stomach projects directly upward with tortuosity of the esophagus; 2. paraesophageal, in which the stomach moves upward alongside the esophagus; and 3. congenitally short esophagus, in which the stomach is within the chest. This classification has been modified by Allison[2] into the sliding or direct for the esophagogastric and short esophagus and the rolling for the paraesophageal.

Various anatomic structures, intrinsic and extrinsic, must function in a very finely coordinated manner to effect sphincteric action at the cardioesophageal junction and in the aged, oftentimes these mechanisms are deranged. The esophagus passes through the crura at the level of the diaphragm and fibers of the right crus encircle at this point to form a sling. The crura are supplied by the phrenic and first lumbar nerves. The left gastric artery has a tethering effect on the lesser curvature near the cardia.

The phrenoesophageal membrane, the intrinsic gastric muscles which form a sling around the esophagus, the intussusception-like propulsion mechanism of the esophagus at the phrenic ampulla, the large gastric rugae which fill the

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opening after ingested material enters the stomach, the oblique entry of the esophagus into the stomach, and the flap-like valve produced by the air bubble are involved in maintaining an effective pinchcock to prevent regurgitation, and upward displacement markedly affects their function. Reflux shortening of the esophagus may be initiated by vagus stimulation, either directly or indirectly, and affect these functions.

That there is a sphincter-like action at the diaphragm is shown during fluoroscopic examination; barium is held up during inspiration but during expiration, when the diaphragm relaxes, barium flows through the cardia into the stomach. This action is also observed during esophagoscopy and at operation.

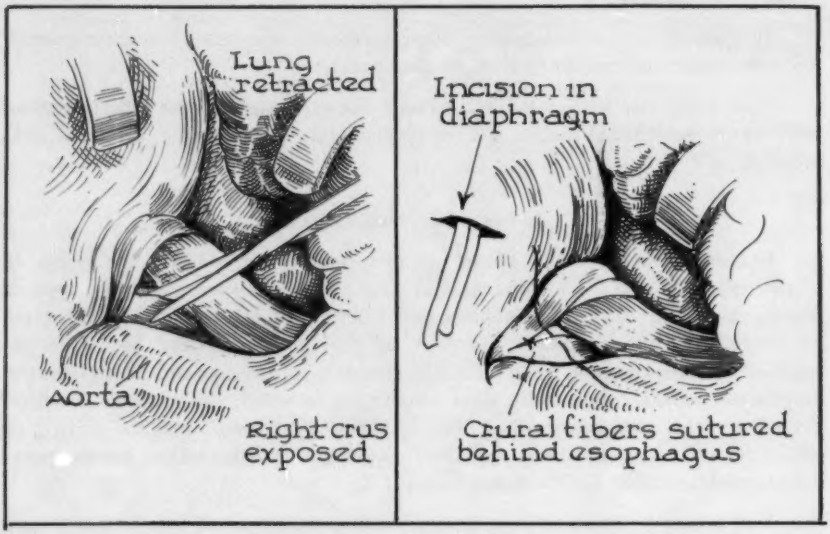

Fig. 1—The technic of Allison is designed to restore the normal anatomic relationship at the diaphragm by closing the triangular defect in the crura behind the esophagus, thus allowing the crura to continue their contractile function.

Esophagoscopy is of importance in the differential diagnosis and is a guide in treatment.

When a hiatal hernia causes severe symptoms in this age group, surgery is indicated.

The hernia can be approached transabdominally or transthoracically. Many patients have associated intraabdominal lesions correctable by surgery. Elderly patients tolerate abdominal surgery better. In addition, an occasional patient is plagued with intercostal neuralgia following the thoracic operation. The same method of repair can be used in either route.

Currently we believe that the thoracic approach in elderly patients should be reserved for those in whom concern exists about the length of the esophagus.

Despite some evidence in this study to support phrenic crush as an adjunct in gastrointestinal hemorrhage whose presumable source is the low esophagus or cardia, the procedure would seem to have little merit. The patient would, in most instances, fare better from a transabdominal approach to establish the source of hemorrhage and carry out appropriate treatment.

Technical Aspects of Restorative Surgery

In repair by the thoracic route, the left hemithorax is entered through the eighth interspace. In the various repairs the phrenic nerve is preserved.

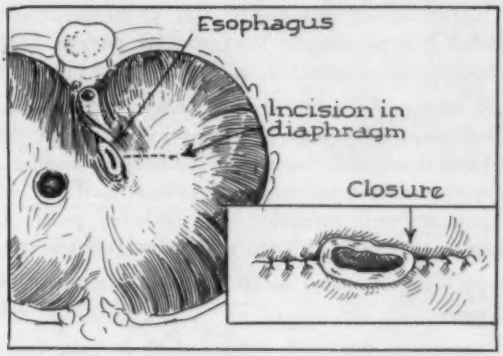

Fig. 2—The technic of Rives and Strug[3] is designed to create a new hiatus by shifting the hiatus laterally.

The technic of Allison[2] is designed to restore the normal anatomic relationship at the diaphragm by closing the triangular defect in the crura behind the esophagus, thus allowing the crura to continue their contractile function (Fig. 1). The oblique entry of the esophagus into the stomach is also restored by tacking the phrenoesophageal membrane and the stomach to the undersurface of the diaphragm.

The technic of Rives and Strug[3] is designed to create a new hiatus by shifting the hiatus laterally (Fig. 2). There has been considerable criticism of the shift because the esophagogastric junction is straightened and esophagitis is reported to be more frequent with this method.

Repair via the abdominal route is accomplished by bringing together the two limbs of the right crus behind the esophagus, exactly as is done through the chest (Fig. 3). Presently an additional step is being carried out: the oblique

angle is restored by suturing the stomach to the esophagus and onto the diaphgram.

Current Study

A total of 121 patients past 60 years of age were seen with esophageal hiatal hernia from 1943 through 1955 at Charity Hospital. The series included 62 women and 59 men. Their ages ranged from 60-93 years. Thirty-nine patients were operated on for hernia; 15 were women and 24 were men.

Nonoperated Cases

Eighty-two patients were treated by nonsurgical methods; 47 were women and 35 were men. Fifty-five associated lesions of the alimentary and biliary tracts were found. In this group 19 patients had colonic diverticula, 14 had cholelithiasis, 7 had duodenal diverticula, and 4, duodenal ulcers. Three had gastric ulcers and 3 had esophageal varices. One patient had an esophageal diverticulum, 1 had jejunal diverticula, and another an esophageal ulcer.

Heart disease was a prominent feature in 48 patients. Various pulmonary affections, the most common being emphysema, crippled 14 patients. Five patients carried residual disabilities from previous cerebrovascular episodes. Portal cirrhosis was encountered in 4 patients. One patient died with carcinoma of the pancreas. Many patients were invalided with diabetes, prostatic and renal diseases, arteriosclerosis, glaucoma, arthritis and other geriatric ailments.

Esophagoscopy was performed in 16 patients. Gross changes of esophagitis were seen in 10 patients.

Patients in this group were not subjected to surgery for several reasons. Medical management was recommended in 54 patients. This regimen was preferred because of "age" in 35 patients. Six patients had minimal symptoms and 7 were asymptomatic. Instrumental esophageal rupture precluded a definitive procedure in 1 patient.

Thirteen patients had more than one associated severe infirmity and operation was not recommended because of the poor general condition. Eight patients refused the proffered surgery and another 3 deserted the hospital. In 2, the hiatal hernia was an incidental autopsy finding.

Operated Cases

Thirty-nine patients whose ages ranged from 60 to 87 years of age underwent 45 operative procedures.

Phrenic Crush

Ten patients had phrenic crush. Two of these were supradiaphragmatic crushes in conjunction with transthoracic hernial repair, and are not considered

here. In 8 patients the left phrenic nerve was crushed 12 times; 5 patients had 1 crush, 2 patients 2 crushes, and 1 patient, 3.

Phrenic crush was considered indicated in 6 patients who were bleeding from the upper gastrointestinal tract. The hematocrit was 22 or below in all 6 patients. The crush was effective for control of hemorrhage in all but one patient who was bleeding from esophageal varices.

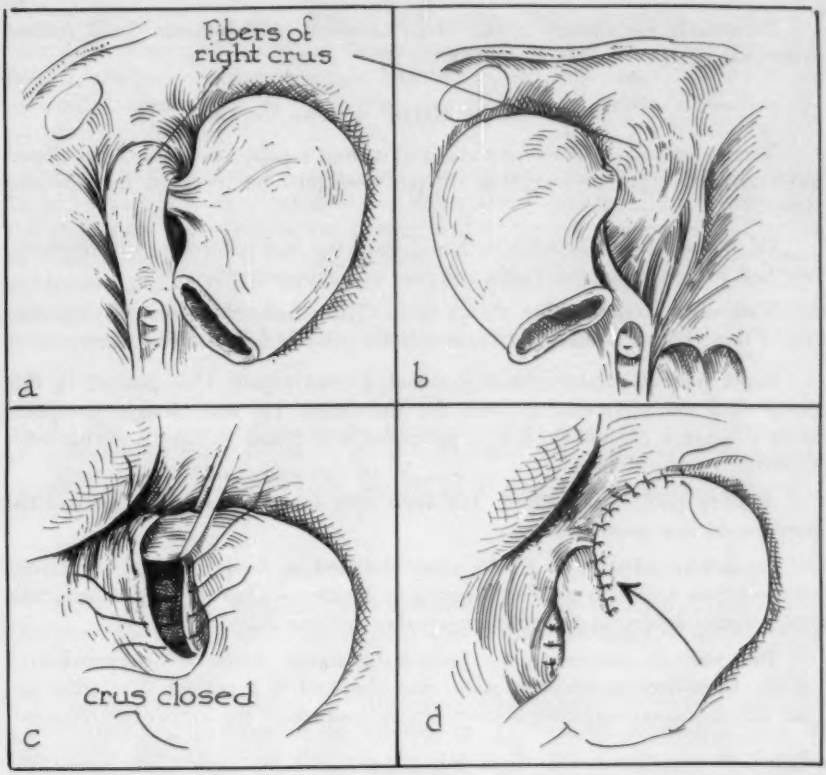

Fig. 3–The oblique angle is restored by suturing the stomach to the esophagus and onto the diaphragm.

Failure to respond to medical measures constituted the indication for crush in 2 patients. In 4, the specific reason for electing crush was not listed.

Four patients who had 8 phrenic crushes developed recurrent symptoms within 3 months. One patient died 4 weeks after crush and was not subject to evaluation. Three patients claimed improvement with a follow-up of less than 6 months.

Transabdominal Hiatal Hernia Repair

Nine patients underwent repair via the abdominal route. Although hematemesis had been a previous manifestation, surgery was carried out electively for other symptoms, usually pain and obstruction.

One patient died from a pulmonary embolus on the fifth postoperative day. The other 8 recovered uneventfully.

Immediate satisfactory results were obtained in 7 patients. In 1 patient symptoms recurred within 6 months.

Transthoracic Hiatal Hernia Repair

Twelve patients underwent a classical Allison repair. One patient developed moderate atelectasis and another, 20 per cent pneumothorax on the opposite side.

Of 3 who were followed less than 1 year, one had recurrence of symptoms, two had proven recurrence, although one was asymptomatic.

Nine were followed more than 2 years. Three had recurrence of symptoms; one of these had a radiologic recurrence. Six patients had satisfactory results.

Eight patients underwent a modified Allison repair. One patient in this group died, probably due to electrolye imbalance. He was thought preoperatively to have a carcinoma, but at operation was found to have a hernia with esophagitis and stricture.

Another patient developed, but recovered from, a moderate mediastinal emphysema and pneumothorax.

Immediate satisfactory results were obtained in 3 patients. Of 4 followed for more than a year, symptoms recurred in 3 patients. One of the 3 patients was subsequently subjected to 2 phrenic crushes without relief.

Two patients underwent the Rives-Strug repair. Atelectasis occurred in 1 patient. Immediate satisfactory result was obtained in 1 patient. The other patient has remained asymptomatic for 9 years, although the hernia has recurred.

Summary

The records of 121 patients past 60 years of age with diaphragmatic hiatal hernia admitted to Charity Hospital, New Orleans, La. during the years 1943 through 1955 have been reviewed.

Eighty-two patients were treated by nonsurgical methods. Fifty-five associated lesions of the alimentary and biliary tracts were present. Other than these 55 lesions, 112 constitutional ailments burdened this geriatric group. Heart disease was a prominent feature in 48 patients. Various pulmonary affections crippled 14 patients.

Transthoracic repair of hiatal hernia was carried out in 22 patients. The oldest patient was operated on at 87. At 91 she was doing well. Twelve patients achieved satisfactory relief of their symptoms. Nine soon had recurrence of their symptoms. One patient died.

Transabdominal repair was employed in 9 patients. Seven got relief with operation and 1 developed a recurrence of symptoms. One patient died.

Phrenic crush was used 12 times in 8 patients. Six crushes were done in the face of massive gastrointestinal hemorrhage and in the single exception of a patient who had esophageal varices, bleeding ceased following this measure. No permanent amelioration of other symptoms referable to hiatal hernia was obtained with this procedure.

Equal anatomic restoration of the crura and the cardioesophageal angle can be accomplished from either above or below the diaphragm. The advantage of the abdominal procedure is that it may lead to the recognition and correction of other intraabdominal lesions. Because most elderly patients have decreased pulmonary function, the abdominal procedure is better tolerated. Intercostal neuralgia is an occasional sequel of the thoracic procedure. For these reasons an abdominal approach is usually advisable in the elderly patient.

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Discussion

Dr. I. Snapper:—Medicine often suffers because our semantics are poor and this certainly holds true in the disease which originally went under the name of cardiospasm.

The esophagus consists of two parts, the tubular esophagus and the gastroesophageal junction or the vestibulum. The tubular esophagus starts at the caudal termination of the pharynx, and ends just above the diaphragm, where the gastroesophageal junction begins. The latter, also designated the vestibulum, is about 3 cm. long. The muscle bundles which delineate the oral and caudal ends of the vestibular part of the esophagus are so weak that they can hardly act like real sphincters. The vestibulum in its entirety, however, acts as a sphincter which in rest is contracted and prevents reflux from the stomach into the esophagus. The vestibulum relaxes only during the last phase of the swallowing act.

In the course of the swallowing act the bolus is forcefully pushed down into the oral part of the tubular esophagus by the combined action of the oral

and pharyngeal muscles. The food is transported through the middle and lower part of the tubular esophagus by a strong "stripping" peristaltic wave of the esophageal wall. During this phase of the swallowing act both the inferior sphincter of the esophagus and the vestibulum are closed. After the bolus has reached the terminal end of the tubular esophagus, the vestibular musculature relaxes as one unit, the vestibulum fills and the food reaches the lumen of the stomach. Therefore, the transport of the food through the esophagus requires *contraction* of the tubular esophagus, followed by *relaxation* of the vestibulum.

The musculature of the tubular and the vestibular parts of the esophagus, also of the two sphincters is innervated by the vagus. The sympathetic nervous system has no influence upon the swallowing mechanism. The same vagus stimulation causes first the contraction of the tubular esophagus, thereafter the relaxation of the vestibular part of the esophagus. Failure of the innervation of the esophagus will therefore bring about both an insufficient contraction or even a paralysis of the tubular esophagus and an absence of the relaxation of the vestibulum.

In so-called cardiospasm the x-rays reveal that the tubular esophagus is tremendously dilated and full of retained food. At the same time, the vestibulum remains contracted and doesn't relax when food has passed through the tubular esophagus.

Hurst clearly proved that the dilatation of the esophagus in so-called cardiospasm is not due to a spasm of the sphincter of the cardia because in such patients a hollow tube, filled with mercury, passes without difficulty through the cardia just as is the case in normal persons. Hurst, therefore, assumed that the main factor which caused the delay of the transport of the food consisted not of spasm but of lack of relaxation of the sphincter of the cardia,—in modern nomenclature of the esophagogastric junction, i.e. the vestibulum, hence the term achalasia.

The slight impediment, caused by this lack of relaxation, however, can never be sufficient to lead to the marked dilatation of the esophagus. As a matter of fact, in achalasia the vagus innervation of the esophagus is damaged as proven by a degeneration of the ganglion cells of the myenteric plexus of Auerbach, situated within the wall of the esophagus. Due to the impaired vagal innervation no peristaltic wave sets in when a bolus arrives at the oral end of the paralyzed tubular esophagus. Thus, the food can only be transported under influence of the force of gravity. When the bolus reaches the vestibulum the vagus-induced relaxation of the esophagogastric junction does not take place and the bolus cannot reach the lumen of the stomach.

It follows that unfortunately the term achalasia is also incomplete, because this name emphasizes only the insufficient relaxation of the vestibulum, but does not mention the paralysis of the tubular esophagus. This has displeased modern

clinicians, well versed in this field who, therefore, have introduced a third term, —megaesophagus which draws an analogy between this disease and the megacolon or Hirschsprung's disease. The latter is also a manifestation of faulty cholinergic innervation of part of the colon due to a degeneration of the myenteric plexus.

The etiology of the degeneration of the myenteric plexus is completely unclear. In olden times, we had the impression that patients who had suffered from diphtheria with postdiphtheritic paralysis were liable to later develop a so-called cardiospasm or achalasia. Since postdiphtheritic polyneuritis often involved the vagus nerve, this would have fitted well in the modern conception that a megaesophagus is caused by a defect of the cholinergic innervation of the esophagus. This concept may well have been correct because recently physicians in Brazil have reported that in Chagas' disease, another ailment with frequent polyneuritis, achalasia of the esophagus is a common sequel.

Nevertheless, diphtheria cannot have been a frequent forerunner of achalasia since many physicians even of riper age have never seen postdiphtheritic paralysis, and achalasia still occurs.

In achalasia, all operations which anastomose the vestibulum with the stomach are useless because they do not improve the paralysis of the tubular esophagus. No wonder that the results of these esophagogastric anastomoses are very poor indeed.

The old Heller operation, transection of the muscle fibers of the cardia, however, is rather frequently successful. Many surgeons add a vagotomy to the Heller operation. Since this results in a paralysis of the stomach, a pyloroplastic must be added.

The vagotomy depresses the acid secretion in the stomach. Therefore, if due to the Heller operation reflux takes place, the fluid which bathes the lower part of the esophagus, is at least not acid and peptic.

We don't clearly understand why the Heller operation is successful in achalasia or why the comparable Rammstedt operation helps in pylorospasm of the infant. This should not, however, gravel us too much. We also do not know why digitalis works in heart disease, quinine in malaria, etc. Since we use medicaments the actions of which are not well understood, why shouldn't we use operations we don't understand,—as long as they are successful.

Dr. O. H. Wangensteen:—Burton wrote, long years ago, in the "Anatomy of Melancholy", "the spleen is full of mystery". What Dr. Snapper had to say about the esophagus in its relationship to the alimentary canal emphasizes the circumstance that there are still a few mysteries concerning the esophagus and its behavior that we do not fully understand.

In his assessment of the problems of benign disease of the esophagus, Dr. Strug leaned rather heavily, I thought, towards substitution therapy for benign stricture. For corrosive strictures following ingestion of lye, I would agree for that type of stricturing process, restoration of satisfactory continuity demands that the stomach, small intestine or the colon be elevated into the neck or upper thorax to replace the obliterated esophageal tube. Moreover, as he emphasized, the colon has the great advantage over use of the stomach, in that it precludes the occurrence of reflux esophagitis. For so-called idiopathic stricture of the esophagus, which in my experience is acid-peptic narrowing of the esophageal tube, owing to reflux of gastric juice—for this stenosing process, the experience of our clinic suggests that gastric resection is the simplest and also an effective treatment.

My first operations for such patients were essentially serendipitous in nature. Horace Walpole, you will remember in the "Three Princes of Serendip", wrote of finding something not being looked for; many a journey, as you and I know, begins and ends like that.

In September, 1939, I operated on a patient for a massive gastric hemorrhage from a duodenal ulcer. He had been dilated in our clinic for more than six years—a hundred times or more for a so-called idiopathic esophageal stricture. I told him, of course, that the operation for massive hemorrhage had nothing to do with stricture and that he should continue to have the stricture dilated. He consented reluctantly twice, insisting that he no longer needed esophageal dilatations. That is now almost 20 years ago; the esophagus now admits a number 46 dilator and he has had no trouble in swallowing.

In 1942, I saw another case. Then for the first time I began to understand what we were dealing with. In the intervening years, we have treated approximately 30 patients with so-called idiopathic esophageal stricture in this manner. It has been a very satisfying experience. My colleague, Dr. MacLean, *(Surg., Gynec. & Obst.*, 103:5, 1956) summarized our experience up to that time of managing acid-peptic stricture, which term has come to supplant the designation, "idiopathic". There has been no mortality; the results have been all that the patient and the surgeon could desire. I wonder what risk Dr. Strug and other surgeons, who find it necessary to resect the esophagus for this condition, transplanting the colon or other hollow viscus as a substitute tube, assume for the patient in this more aggressive procedure.

Nakayama of Chiba City, Japan, places the stomach subcutaneously to meet the esophagus just above or below the clavicle, when excising the esophagus for cancer. In that way Nakayama gets away from the greater risk of an intrathoracic anastomoses. In adults suffering from cancer of the esophagus, subcutaneous placement of the esophagus is acceptable. For children who are justifiably sensitive about their appearance, surgeons, who have completed restoration of alimentary tract continuity by bringing up a jejunal loop sub-

cutaneously for correction of congenital esophageal atresia, have usually found it necessary to divide the bony thorax subsequently to permit placement of the tortuous jejunal loop inside the thorax.

I have supplanted the esophagus for corrosive strictures (lye and sulphuric acid) by elevating the stomach into the thorax or neck. One such man had undergone more than 600 dilatations when he finally asked to have the stenosed esophagus removed by a cervical esophagogastric anastomosis. For seven years, he was perfectly well. Recently we have found it necessary to dilate him occasionally—a circumstance which suggests the superiority of using the colon rather than the stomach, as Dr. Strug has done it.

To preclude the occurrence of acid-peptic stricture, following excision of esophageal strictures, my erstwhile colleagues, Drs. H. W. Clatworthy of Columbus (*Surgery* 36:399, 1954) and K. A. Merendino of Seattle (*Ann. Surg.* 141:201, 1955) suggest interposition of a jejunal loop between the esophagus and the stomach to get around the stenosing influence of direct contact between stomach and esophagus, when the cardiac sphincter of the esophagus has been sacrificed. Dr. David V. Habif (1958) of New York tells me he performed such an operation in 1952. He points out too that these are variants of the Roux-Y procedure.

Concerning the conventional Heller procedure, I have had some misgivings over its accomplishment in overcoming the retention of a dilated tortuous megaesophagus.

My first reaction in seeking a more adequate procedure for megaesophagus was to excise the entire acid-secreting area of the stomach, anastomosing the antrum to the esophagus after straightening and shortening it. That has proved an adequate operation. Those patients too, however, develop a megaloblastic anemia as do patients who accept total gastrectomy for gastric cancer. I submitted seven patients to this procedure. They maintain their health by taking Vitamin B_{12} intramuscularly once a month. It is indeed interesting that loss of the acid-secreting area produces megaloblastic anemia, despite the presence of the antrum.

This experience taught me to have another look at the Heller procedure. In contemplating failure of that operation to effect complete emptying of the esophagus, I began to employ a balloon at the time of the Heller operation to help disrupt the remaining muscle fibers of the esophageal wall, which the conventional Heller leaves intact. When one has completely disrupted the esophageal muscle fibers in this manner when operating for megaesophagus, he will find, almost invariably, the esophagus empties completely and directly. I have one patient who had suffered from cardiospasm for 55 years. The esophagus held three and a half quarts of barium; it had a number of sigmoid curves. Save where the sharp S curves hold up the barium column, the remainder of the

esophagus empties completely and directly. I agree with Dr. Snapper one should make a pyloroplasty. I see no need for vagotomy when one is operating for cardiospasm. Pyloroplasty insures ready empyting of the stomach.

With reference to Dr. Craighead's paper, I like his approach to this problem. I, too, correct hiatal hernia from below. It is interesting that here in New Orleans too, a group of surgeons has performed lateral migration of the esophagus as Drs. Merendino, Varco and I did, a few years ago (*Ann. Surg.* 129:185-197, 1949). I find that by employing an extrapleural split of the lower sternum one has ready access to the esophageal crura. By adding pyloroplasty to this correction, one has a very acceptable operation with which to deal with hiatal hernia.

Dr. Craighead spoke of so-called congenitally short esophagus. Norman Barrett has interjected a new idea by indicating as Dr. Craighead showed us that the lower end of the esophagus may be lined by gastric epithelium. Norman Barrett, somewhat facetiously, says therefore that the congenitally short esophagus is not short.

In fact, I have never seen the stomach within the thorax in so-called congenitally short esophagus. It has always been below the diaphragm. English surgeons, who deal with conditions of the esophagus like our own American thoracic surgeons, usually do their own endoscopic procedures. It is not unusual to see Norman Barrett or Norman Tanner start off their program of operations by doing three or four endoscopies before they address themselves seriously to their day's work. If they see a fiery red mucosa in the lower esophagus, they know they may be dealing with the kind of congenitally short esophagus that Dr. Craighead spoke of.

I should like to ask Dr. Craighead if he has ever seen a congenital short esophagus in which the stomach is in the thorax, such as Percival Bailey (1919) described in the dissecting rooms a number of years ago, while still a medical student.

There are a number of other conditions of the esophagus bearing upon these interesting papers which merit discussion, but I have said enough. The hour is late. I would like to say here again: in a way, I am like the preacher pronouncing the benediction. He finds it necessary to do it every Sunday. The things I have said today, from this platform, I have said to this audience on other occasions. Let me add one final word concerning so-called spontaneous perforation of the esophagus: experimental evidence suggests very definitely that spontaneous perforation of the esophagus is acid-peptic perforation of the esophagus. If one excises the entire acid-peptic secreting area of the stomach, in a dog, obstruction of the pylorus does not injure the esophageal mucosa, even though the force of vomiting remains the same.

Dr. Lawrence H. Strug:—There are just two additional comments I would like to make.

Our series of esophageal substitution, which include, both the colon, jejunum, and stomach is not very large. In the 13 cases of jejunal and colon substitution for benign disease, there were no deaths. It is likely that as we begin to do them more frequently our mortality will catch up with us. I am sure it will.

In reply to the question "When is the proper time to begin dilatation in acute corrosive esophagitis?" There is some controversy over this point. On our service we usually wait until the acute symptoms subside, anywhere from five to ten days or two weeks. Others, however, think differently and the literature is full of differences of opinion, as to whether or not early dilatation is beneficial.

As to complications occurring with early intubation in corrosive esophagitis, here again I wish to state that on our service we do not use early intubation. In former years early intubation was practiced on the EENT service. A number of perforations of the esophagus occurred following esophagoscopy.

Dr. Claude C. Craighead:—In answer to Dr. Wangensteen's questions, I have not seen a patient with a congenital short esophagus and believe that it is a very infrequent occurrence.

The drawings shown in discussion of our paper were taken from Norman Barrett's work on hiatal hernia. As to how frequently hiatal hernias occur in pregnant women, the figures range from 10 to 25 per cent. The most common type is the direct hernia.

How often is surgery for hiatal hernia necessary? Actually we have about a 2-4 per cent, I think, who undergo surgery. In a previous study I reviewed 250 symptomatic radiologic hernias. We had only five operations during that particular phase, so we see a large number of small diaphragmatic hernias that do not ever come to surgery.

LESIONS INVOLVING THE ORAL MUCOSA*

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The alimentary canal is the "Alpha to Omega" of the gastrointestinal tract, with the mouth and its contained structures the "Alpha" end. There is probably no portion of the human body that takes the abuse and contains the multitude of normal and pathogenic microorganisms as that found in the oral cavity. It is no wonder then that more diseases both local and systemic can be detected in the mouth than any other portion or system of the human body.

In considering the great number of lesions in the oral cavity we might classify them under definite heading as to their character and origin as follows:

1. Trauma of food, teeth deposits, artificial dentures or prostheses or occupational conditions.
2. Local infections involving teeth and supporting structures, caused by bacteria or fungi.
3. Irritations of metals or metallic poisons, including radioactive agents.
4. Allergies.
5. Dermatoses.
6. Deficiency diseases and nutrition.
7. Blood dyscrasias.
8. Salivary gland dysfunctions.
9. Oral manifestations of systemic diseases.
10. Neurotrophic disturbances.
11. Thermal, chemical and electrical irritations.
12. Neoplastic diseases.

The mouth, which is normally the port of entry of all of our nutritional intake, is subjected to a variety of temperature changes and to the trauma of a great many types of raw foodstuffs that are subjected to the mechanical attack of the efforts of the teeth to reduce these foods to a state where they can be broken up and prepared for the initial digestion which starts in the mouth and sends them on their way through the long and tortuous journey through the gastrointestinal tract. Where teeth have been lost and have been replaced by

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prosthetic appliances we often have the trauma of these substitutes to deal with especially where the physiological absorption of the supporting structures have rendered these appliances ill-fitting in view of the changes which have taken place. This often results in irritative hypertrophy of the oral mucosa and its underlying connective tissue.

The simple lesions of the oral mucosa have been grouped under the general heading of stomatitis. The mucous membrane covering the gums is that part most frequently first to be affected and is generally spoken of as a gingivitis. The deficiency diseases such as avitaminosis of scurvy give rise to an inflammatory condition characterized by bogginess of the gums with marked hyperemia and bleeding of the gums with the accompanied loosening of the teeth. Fortunately this condition is now rare in this country because of the availability of Vitamin C or ascorbic acid in our modern diets.

Some of the metabolic diseases such as diabetes have typical manifestations about the gingiva and the supporting structures of the teeth. The gingiva in uncontrolled diabetics is usually a deep red color and the tissues have an edematous appearance with some enlargement. The patient may complain of dryness or burning of the tongue with hyperemia of the fungiform papillae. There is often involvement of the supportive tissues of the teeth with loosening and early loss of teeth due to peridontoclosia or pyorrhea.

Among the lesions of the oral mucosa which give us a great deal of concern are the various blood dyscrasias which are quite numerous. The anemias are varied, the simpler anemias are characterized by an atrophy of the mucosa of the tongue accompanied by a burning sensation, with a pale appearance of the tissues. In pernicious anemia there is often a painful glossitis of a fiery red color which may later become smooth and feel stiff and may even have a loss of taste.

In aplastic anemia the first symptom may be a bleeding of the margin of the gums with marked swelling. This may go on with remission of the oral symptoms.

Agranulocytosis or agranulocytic angina may be characterized by sudden appearance of ulcerative bleeding of the mucosa which may go on to a localized sloughing area. This condition used to be precipitated by drug reactions such as the use of Amidopyrene or by the use of some of the sulfa drugs. Since the cutting down of the use of these drugs this disease is not commonly seen.

Mononucleosis is one of the blood dyscrasias which is characterized by hemorrhages into the palate. This frequently can be diagnosed by the small petechial hemorrhages especially in the area of the soft palate and uvula.

Thrombocytopenic purpura may be characterized by the sudden appearance of large pupuric spots beneath the mucosa and the gingiva frequently bleeds due to the trauma of these tissues due to eating.

Acute leukemia is often characterized by a gingival enlargement which has a marked venous engorgement with discoloration of the tissues. The enlargement may even cover some of the teeth at times, and tends to ulcerate and bleed spontaneously. Any surgical trauma under these conditions such as the extraction of teeth may lead to a fatality within hours or a few days. It is important in any acute bleeding hypertrophies of the gums to do an immediate complete blood count and differential count to make a diagnosis of this condition if surgery has been contemplated.

In *polycythemia vera* there is a very marked red color of all the mucous membranes of the mouth and tongue but rarely any bleeding. This condition can be readily diagnosed because it is always associated with hyperemia of the vessels of the eye and a marked reddening of all the skin.

Sprue or macrocytic anemia has at times affected the mucosa of the mouth in that the tongue may be sore and tender with a beefy red appearance and with hemorrhages of a purpura nature in the mucous membranes. This condition is often associated with a Vitamin B_{12} and folic acid deficiency.

There is another group of diseases which shows typical manifestations in the mucous membrane covering the gingiva. This is that group of metallic poisons which have a particular affinity for depositions in the margins of the gums. The gray line of plumbism or lead poisoning is typical of this. Workers in industries using lead in various forms such as painters, storage-battery workers, and lead smelters are often seen with a gray line of deposit a short distance away from the gingival margin of the gums which under magnification shows distinct dark granules making up the "lead line".

Mercuralism from the mercury compounds used in the treatment of syphilis and in the mercurial ointments used in various dermatoses may result in a gingivitis or mercurial stomatitis which may go on to sloughing of the oral mucosa and involvement of the underlying tissues.

Arsenical compounds used in industry have found their way into the mouth and supporting tissues of the teeth resulting in an arsenical necrosis. Phosphorus also has a similar effect on the gingival tissues. Bismuth and silver also produce a pigmentation of the gums while some silver salts produce an orgyria of all the surface tissues of the body.

The dermatological diseases manifested in the mouth are quite common. The simplest of these is the appearance of the apthous lesions or ulcers which occur on the mucosa of the lips, cheeks and tongue. These are transitory lesions of small surface ulcerations which often are of short duration with a tendency to recur. These lesions are sometimes associated with emotional instability or may be associated with a dietetic sensitivity. They are also thought by some to be of gonadal origin. These lesions have responded in some cases to injections

of gamma globulin. Estrogen therapy has also been used in some cases with a degree of success. Personally I have had excellent results in the individual ulcerations by treating the lesion with a simple topical application of a saturated solution of salicylic acid dissolved in alcohol. One application generally clears up the ulceration but it does not prevent other lesions from recurring.

Herpes simplex is a vesicular eruption frequently seen about the lips or oral commissures but rarely occurring in the mucosa of the mouth. These lesions are thought to be of a virus origin. The vesicules rupture early and have a yellow exudate which results in an unstable scab. These lesions are usually treated with mildly caustic agents such as silver nitrate, trichloracetic acid, alum or by such drugs as tincture of benzoin or by some of the antihistaminic agents. The duration is often reduced by large doses of Vitamin B-complex with Vitamin C.

Thrush or moniliasis is one of the infections seen on the mucosa of the mouth or tongue. This is a yeast-like fungus known as *Candida albicans*. The disease is more frequently seen in children especially nursing infants. It is more common in institutions where the hygienic conditions are bad. It is also seen in people wearing dentures which are not properly cleaned. It is not uncommon today in adults who have been in the habit of using antibiotic troches in the mouth. This is due to the disturbance of the normal flora of the mouth where the fungi, which are usually controlled by the bacterial cocci, are stimulated to abnormal growth when these cocci are destroyed by the broad-spectrum antibiotics. These fungus infections are characterized by irregular denuded areas of the tongue, cheeks or palate. Pearly white lesions of the surface are present and are removed with difficulty. They are attended by pain in the mouth and throat. A differential diagnosis can be made by the examination of smears or cultures taken from the areas. The treatment consists in the local irrigations of solutions of Mycostatin and the discontinuance of the use of the broad-spectrum antibiotics locally. Topical applications of 1 per cent Crystal violet solutions have frequently been effective.

Lichen planus is another dermatological lesion seen occasionally on the oral mucosa. These lesions appear as diffusely outlined bluish-white raised hyperkeratinized areas which have a string-like pattern with a reticular distribution often interlacing. A large central plaque lesion on the tongue may be present. The origin of these lesions are vague but it is thought by some to be associated with an emotional or neurotrophic disorder. It does not readily respond to any specific type of therapy.

Leukoplakia or leukokeratosis is one of the lesions frequently seen in the mouth. It occurs most often in the male after the age of 50 years. It is being seen more frequently now in the female because of the increase of the use of tobacco in women. This disease appears to originate in the mouths of those subjected to the irritating factors such as the uses of very hot fluids, the habitual

use of strong condiments, irritation from broken down or rough teeth, and especially the chronic use of tobacco, more so in the form of the pipe or chewing tobacco. The typical appearance of leukoplakia is a firm white raised patch on the surface of the mucosa which is irregular in outline and is usually concentrated in patches in a localized area. The area is highly keratinized and does not peel off on manipulation. The area is rarely painful and the patient is oblivious of its presence unless he detects it by observation in a mirror or his attention is directed to it after it was observed by his dentist or physician on examination of his mouth. These lesions are persistent and do not readily disappear. Repeated irritation of these lesions may result in a malignant change ending in a squamous cell carcinoma. Because of this the leukoplakia lesions are regarded potentially as one of the precancerous lesions even though the percentage that do develop into a malignancy is small. A well defined and raised area should be biopsied to detect early malignant changes and often the area should be removed by dissection or by the use of an electrocautery. The patient should be warned to avoid the excessive use of tobacco, condiments and extremely hot food and drinks. He should also avoid the continued irritation from teeth or ill-fitting dentures.

Another lesion quite common in the mucosa of the cheeks is the Fordyce Spots or the deposition of the multiple fat granules in the surface of the mucosa. These present minute yellow spots close together in the cheek and are of no pathologic or clinical significance, but may alarm a patient when he observes them.

There are several specific infectious granulomata seen in the oral cavity, among these are syphilis, tuberculosis, actinomycosis, blastomycosis, histoplasmosis, and leprosy. Of these special infections syphilis is the lesion more commonly seen. Acute syphilis is manifested by the presence of a chancre on the lips or oral mucosa. This is characterized by a shallow ulceration with a grayish-white encrusted exudate and raised borders of the ulceration, and an indurated base. This lesion is not very painful as in other ulcerations. The secondary lesion or mucous patch is also typical. The oral commissure is a frequent site. They appear as grayish-white lesions surrounded by an erythematous base. Any trauma of these patches leaves a raw bleeding surface. These patches may also be located on the tongue, the inner surface and vermillion borders of the lips and the pharyngeal tissues. The diagnosis is made by the serological examination or by smears from the ulcers examined under the special technic with the dark field illumination, which may show the *Treponema pallidum*.

Tuberculosis is not a common lesion in the mouth even when the pulmonary tuberculosis is present. It has been found in the mucosa of the tongue, cheek, lips and palate. These lesions start as small tubercules which break down to form a painful ulcer with a tendency to form adjacent ulcers. The lesions become quite chronic and do not readily yield to local treatment. The examination

of the biopsy specimen and the detection of the acid-fast organisms in smears and stained sections makes the diagnosis and differentiates it from chancre, gumma and carcinoma.

Pemphigus is one of the uncommon dermatoses of the oral mucosa which is characterized by the formation of bullae or vesicles. Its origin is uncertain although it is thought to be caused by a virus infection. The lesions as seen in the mouth are painless large vesicles or bulla which appear yellow because of the fluid content and bacterial contamination. These rupture and are replaced by a hard yellow crust. The patients have loss of weight, nervousness and often a pruritus. The diagnosis is made by the history and the histological examination of the bullae. The treatment by ACTH or cortisone has aided in the temporary remission of the symptoms.

One of the acute bacterial infections which involves the oral and pharyngeal mucous membranes is that condition known as Vincent's infection or Vincent's angina. This was popularly called "trench mouth" following World War I when so many soldiers in trench warfare became infected with this disease. This condition is an ulcerative stomatitis caused by the Vincents Spirila and the fusiform bacillus which grow in symbiosis. This disease is characterized by ulcerations of the gingival margins of the gums and the interdental papillae. It is frequently found in young adults about the partially erupted mandibular third molar or "wisdom teeth" as they are popularly called. It also may involve the mucous membrane of the cheeks and pharynx in the acute stages. It presents a gray sloughing gangrenous margin of the gums which bleeds freely on brushing or by contact with food. It often is associated with a foul fetid odor. The gray slough can be removed by irrigation or sloughing with oxidizing washes such as peroxide of hydrogen or sodium perborate. The modern antibiotics such as penicillin in large doses seem to be more or less of a specific in the treatment of this condition. In fact any of the drugs used in the treatment of syphilis may be a specific in its eradication when coupled with good oral hygiene. Experience has shown that smoking has a direct effect in retarding the treatment of this condition.

In discussing the lesions of the oral mucosa, we should include those vague conditions which are noted under the heading of allergies. The response of some individuals who are sensitive to certain foods, drugs and foreign proteins and pollens is often very marked in that the mucous membranes of the vascular areas with loose elastic or areolar tissue are prone to react to specific allergies. Angioneurotic edema is a typical example of the complex symptoms about the mouth, lips and loose tissues of the face. The mucous membranes become engorged and swollen. It may come on very suddenly with marked facial disfigurement with swelling of the tongue, lips and pharynx. In cases involving the mucosa of the glottis it may result in asphyxia unless heroic measures are immediately instituted. Most of these allergic reactions, however, respond to administrations of

adrenalin or to the antihistamines. The identification of the specific agent which brought on the allergic reaction should always be attempted so that the patient should be immunized to avoid future episodes.

Many of the xanthematous diseases of children have distinct manifestations in and about the oral mucosa. Chief of these are the characteristic Koplic spots of Rubeola or measles. These spots are important in the diagnosis of measles as they occur one or two days before the onset of the typical rash which appears on the skin surfaces of the body. The Koplic spots appear on the cheek opposite the molar teeth as tiny white popules surrounded by an area of inflammatory areola. This inflammatory area may spread to the laryngeal and tracheobronchial mucous membranes.

Scarlet fever is accompanied by a uniformly congested mucosa of the cheeks and tongue. Fine submucous hemorrhages often on the palate and the margins of the tongue and the development of hyperemic fungiform papillae appear as bright red spots which give use to the term "strawberry tongue". In past years in debilitated individuals there was occasionally seen a severe stomatitis following scarlet fever which resulted in a rapidly fulminating necrotic area known as *noma* or *chancrum oris*. This condition now is extremely rare due to better mouth hygiene and the immunization of children shown to have a susceptibility as indicated by the Dick test.

Diphtheria is another infectious disease that has a particular affinity for the mucous membranes of the mouth and pharynx. Its particular area is generally in or about the tonsils. This disease is often spread by the nasal or throat discharges of carriers who themselves are immune or show no clinical symptoms of the disease. Diphtheria is characterized by an angina with the development of a pseudomembrane involving the pharynx tonsils and often the larynx. This membrane is of a dirty gray color and is not readily detached and is associated with edema and hyperemia of the adjacent tissues. The diagnosis is readily made by the clinical symptoms and by examination of the cultures made of smears taken from the area as well as confirmation by the Schick Test. Diphtheria is not as common today as formerly because of the almost universal immunization of children by diphtheria antitoxin and by later booster doses of toxoid.

The oral mucous membranes are the site of numerous benign lesions or neoplastic growths which are occasionally seen. The simplest of these are the small pedunculated papillomata which occur about the cheeks and tongue. Some of these are of obvious traumatic origin started by biting the tissues. There are other cornifying types which might be associated with senile tissue changes.

Hemangiomas or varicosities may be present in a variety of locations in the mouth but most often appear on the cheeks or tongue. These vascular enlargements rarely attain any great size and may be controlled by injecting the

area with sclerosing solutions or by electrocoagulation, and should be so handled if they are where the tissue may be traumatized by the teeth.

Oral cancer is of course a great concern to all of us, but fortunately it is in a position where it may be visualized by routine examination of the mouth by a dentist or a physician. About 5 per cent of the patients who die of cancer in the United States have lesions which had their origin in the mucous membrane of the oral cavity. The etiology of most oral cancers are irritations that persist over a long period of time, such as the habitual use of tobacco in pipe smokers or tobacco or snuff chewers. The irritation of badly carious teeth and ill-fitting dentures, the long exposure of the mucosa of the lips to the actinic rays of the sun as occurs in farmers and fishermen in Southern climates accounts for many of the initial lesions in this area.

Ninety per cent of the cancers about the oral mucosa are of epidermoid or squamous types of carcinoma. The areas most frequently involved are in the following order: 1. lip; 2. tongue; 3. tonsil; 4. gums; 5. hard palate; 6. soft palate; 7. floor of the mouth; 8. buccal surface of the cheek. This order of frequency is from the records of the Ancologic Hospital of Philadelphia.

The gross appearance of the carcinomas in the mouth is usually a nodular growth with an irregular surface tending to fungate. The margins are usually firm with the base indurated and firm. Early ulceration of the surface or a tendency to form a crater is not usually a sign of carcinoma. The growth is usually outward, but this is not of itself an indication of its degree of malignancy. Those which have a downward growth which infiltrate into the basal tissue are more apt to be more malignant especially where there is an invasion of the surrounding lymph nodes. The carcinomata of the lower portion of the mouth below the level of the occlusion of the teeth is apt to metastasize much more readily than those occurring in the upper half of the mouth.

The predisposing lesions in the mouth that may undergo malignancy are leukoplakia, syphilitic glossitis, papillomata, senile keratosis, seborrhoic changes in the above lesions should warrant an immediate biopsy or an excision with an immediate frozen section biopsy. In this way many incipient malignant lesions may be treated before they infiltrate and metastasize and a permanent cure may be accomplished which will save the life of the patient.

Discussion

Dr. O. H. Wangensteen:—It never occurred to me until recently that I would ever have a serious interest in teeth beyond my own. Dr. Parker talked about the mouth as a mirror of health. John Hunter, great surgeon of his day, was not above thinking of the teeth. In fact, he wrote an excellent monograph on the subject.

The mouth has squamous gingival epithelium. In prior sessions of this group I have indicated how sensitive squamous esophageal epithelium is to injury by digestive juices, especially so to the pepsin of the gastric juice. It strikes me that the problem of gingival erosion is essentially an enzymatic erosive process. Just what the enzymes are that can bring such effects about is not apparent—probably bacteria play an important role.

The medical profession must begin to direct more serious attention to the problem of the teeth and the gums. A tenth of all medical expense in this country, it has been said, goes to pay for problems of dental health. Some organic acids apparently are more active in eroding enamel and dentine than are mineral acids, such as HCl. Enzymes such as phosphatase may play an important role in initiating tooth decay. Bacterial enzymes and the saliva are undoubtedly two important components in the occurrence.

Dr. I. Snapper:—Fortunately we are nowadays hardly ever able to study scurvy which was mentioned by Dr. Parker, because this disease disappeared from our part of the world as soon as the Irish potato became a popular nutrient.

Dr. Parker discussed the glossitis which several decades ago frequently occurred in pernicious anemia. Everything which once upon a time was written in a textbook will be reprinted for 50 years—even if the disease disappears. This holds true for the glossitis with atrophy of the mucous membrane of the tongue in pernicious anemia.

This glossitis together with the fissures at the corners of the mouth, were due to poor absorption of vitamins other than Vitamin B_{12} or cobalamine. In other words, in a patient with pernicious anemia (or another anemia) the glossitis can be cured by the administration of large amounts of Vitamin B_1 to B_6 which as you know do not cure the pernicious anemia. The modern emphasis on vitamin-rich diets must be the reason why nowadays the patients with pernicious anemia do not suffer from an atrophic glossitis anymore.

Already prior to the introduction of the liver therapy, atrophic glossitis in pernicious anemia was unknown in certain areas, especially Russia. This may possibly have been connected with special food habits which perhaps increased the Vitamin B content of the Russian daily diet.

As soon as any denudation of the epithelium of the oral cavity takes place, irrespective of the cause, a secondary infection with the salivary spirochetes occurs. In mercurial stomatitis, in *lupus erythematosus*, in *lichen planus*, in *stomatitis aphthosa*, in Plant-Vincent's angina, in leukoplakia and many other diseases of the mouth, the greater part of the ulcerations are due to this secondary spirochetal invasion and not to the original disease. If the secondary infection is combatted, the lesions improve so much that seemingly a "cure" of the original disease has been obtained.

In olden times arsenicals, especially arsphenamine derivatives were used for this purpose; nowadays penicillin is given. It is true that penicillin kills many spirochetes, but it seems to me that penicillin is less active against salivary spirochetes than against *Treponema pallidum*. As per today, it is difficult to get any arsphenamine preparation in New York, and most pharmacists answer, "Don't you know that for the last 20 years arsphenamine has been replaced by penicillin?" There are, however, pharmacists in New York who still carry one such preparation to wit: stovarsol. One tablet of stovarsol should be crushed and suspended in water. The patient should hold this suspension for one minute in his mouth and then spit it out again. The stovarsol kills the spirochetes and practically no arsphenamine is absorbed into the body. In this way a remarkable improvement can be obtained if in the oral diseases mentioned above excessive ulceration has taken place.

CONGENITAL ANOMALIES OF THE GASTROINTESTINAL TRACT*

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Nearly half of the deaths in pediatric surgery are due to congenital anomalies of the gastrointestinal tract in spite of the fact that these abnormalities are relatively uncommon. There are several factors which unfavorably influence the prognosis of these patients: in the first place, infants with serious anomalies are often born prematurely, and second, since most life-threatening abnormalities originate early in fetal life when all organ-systems are developing, there is a high incidence of multiple anomalies. Third, delay in diagnosis materially increases the risk by adding dehydration and electrolyte depletion to the problems of a patient who must undergo major surgery.

Atresia of the Esophagus

One of the earliest anomalies encountered, in respect to both embryologic origin and symptomatology in postnatal life, is atresia of the esophagus with or without tracheoesophageal fistula. In the third or fourth week of fetal life, the lung bud begins to form on the ventral surface of the primitive fore-gut. If the separation of the respiratory system from the alimentary tube progresses abnormally, the esophagus may be divided, but either one or both segments may remain connected to the trachea as a tracheoesophageal fistula.

In the most common type of this anomaly, the proximal esophagus ends as a blind pouch in the upper chest while the distal esophagus joins the trachea just above the bifurcation. In the absence of a patent esophagus, there is difficulty in resuscitation at birth, excess salivation and intermittent cyanosis, and asphyxia on feeding, due to aspiration of amniotic fluid, saliva, and milk into the trachea and lungs.

The diagnosis of esophageal atresia is suspected when a catheter cannot be passed through the esophagus and is proved when x-rays with a small amount of contrast medium visualize the site of obstruction. The x-rays should always include the abdomen, because absence of swallowed air below the diaphragm suggests agenesis of the distal esophagus.

The diminished cough reflex in infants plays an important role in the pre- and postoperative care of all newborns but particularly those with atresia of the esophagus. If this diagnosis is made before significant aspiration pneumonitis occurs, operation should be undertaken immediately, but if there is extensive

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pulmonary involvement, surgery should be postponed 24 to 48 hours. During this time the nasopharynx is aspirated frequently to remove the secretions in the upper respiratory passages and to stimulate coughing. Other preoperative measures include antibiotics and correction of fluid and electrolyte deficit, which is minimal in esophageal atresia since there is no abnormal loss.

Surgical repair consists of closing the tracheoesophageal fistula and establishing continuity of the esophagus. In closing the fistula, extreme care must be used to prevent stenosis of the tiny trachea, and in repairing the esophagus, excessive tension must be avoided by adequate mobilization.

Postoperatively, emphasis is placed on frequent aspiration of the nasopharynx and avoidance of overhydration. Newborn infants without abnormal loss of fluid require very small amounts of intravenous fluids. Dehydration itself is not thought to harm these patients, but overhydration is often fatal.

The most common cause of death in patients with atresia of the esophagus is aspiration pneumonitis, but the high incidence of other anomalies and of prematurity also contribute to the high mortality of this condition.

Rupture of the Stomach

The embryologic etiology of deficiency of the gastric musculature is not known. The symptoms of this condition result from the peritonitis produced by rupture of the weak area of the stomach. It should be suspected in a newborn infant who becomes distended with or without vomiting and who is demonstrated by x-ray to have pneumoperitoneum. Perforation of the stomach in the neonatal period may also result from trauma of a misdirected tracheal catheter, from a lavage tube, from acute peptic ulcer, or from improperly administered intragastric oxygen.

Demonstration of pneumoperitoneum in a newborn infant is indication for surgery without further investigation. Conservative treatment should not be used, since the tears are often large and since the omentum of the newborn is ineffective in sealing the perforation. As soon as the condition of the patient will permit, the abdomen should be explored and the perforation closed, usually after debriding the necrotic edges or the portion deficient in muscularis. Multiple perforations should be sought and closed separately.

Death in these patients is usually due to peritonitis or overwhelming sepsis and the mortality rate is high. Here again, early diagnosis should reduce the mortality by decreasing the amount of soiling of the peritoneum and by shortening the period of preoperative debilitation.

Intestinal Atresia and Stenosis

Congenital stenosis and atresia of the intestine occur most commonly in the ileum, probably because this is the longest segment of the gastrointestinal tract.

Per unit length, they are most frequent in the duodenum, and there is a high incidence of Mongolism in patients with congenital duodenal obstruction.

During the second month of fetal life the intestine temporarily becomes a solid cord with no lumen. When vacuolization and re-establishment of the lumen occur, interference with development may result in complete or partial intestinal obstruction at one or more sites.

Congenital complete intestinal obstruction is always manifest within a day or so after birth by vomiting with or without abdominal distention. There may be absence of distention in a high obstruction because the entire obstructed segment may be emptied by vomiting. In obstruction distal to the ligament of Treitz, distention may be so massive as to interfere with respiration.

Since meconium is formed throughout the entire length of the intestine, the infant with a high congenital obstruction may pass an almost normal quantity of meconium at the usual time. The stools may be grossly normal, but microscopic examination will reveal the absence of lanugo hair and cornified epithelial cells if the obstruction is a complete one.

It should be emphasized that the appearance of bile-stained vomitus in a small baby is indication for immediate investigation, and should never be considered functional. It may result from a sepsis or from intracranial damage, but is most commonly due to intestinal obstruction below the ampulla of Vater. Investigation for intestinal obstruction consists solely of plain x-rays of the abdomen. The presence of one or more abnormally distended loops of intestine constitute adequate indication for exploration. X-ray studies with contrast material are specifically contraindicated in complete obstruction in infants as well as in adults; the delay in definitive treatment is unjustifiable and during the course of the examination the patient will often vomit and aspirate gastric contents.

Dehydration and electrolyte imbalance must be corrected before surgery and the gastrointestinal tract should be thoroughly decompressed by nasogastric suction. Surgical treatment consists of by-passing a duodenal obstruction or resecting an obstructive lesion below the ligament of Treitz. Since congenital anomalies are so often multiple, the entire gastrointestinal tract should always be examined to rule out the presence of other sites of obstruction. Operative decompression of the obstructed segment will aid materially in closing the abdomen and will improve respiration by reducing the pressure on the diaphragm. Intravenous fluids may have to be continued for several days, because return of effective peristalsis is very slow in the grossly dilated and hypertrophied segment just above a congenital obstruction.

Annular Pancreas

Annular pancreas also has its origin in the second month of fetal life. If the two anlage of the pancreas develop so as to encircle the second portion of the

duodenum, complete or partial obstruction may result. Complete obstruction due to annular pancreas will produce symptoms immediately after birth and will be indistinguishable preoperatively from atresia of the duodenum, but partial obstruction may not be manifest until adult life. When the annular ring of pancreatic tissue causes difficulty, the obstruction should be relieved by duodenoduodenostomy or duodenojejunostomy. Division or resection of a portion of the ring may result in pancreatic fistula.

Imperforate Anus

The normal human fetus at seven weeks has an imperforate anus with a rectourogenital fistula, but normally the fistula (which represents the cloacal duct) migrates posteriorly and inferiorly to the normal location on the perineum surrounded by the external anal sphincter. Incomplete migration will result in imperforate anus with an associated fistula to the bladder, urethra, vagina, or perineum. The imperforate anus can be diagnosed at birth by simple inspection of the perineum, but the associated rectourogenital fistula may not be demonstrated before surgery.

A patient with imperforate anus need not be operated upon until the distal rectal pouch is distended with air, as demonstrated by x-ray, and these same films are used to indicate the distance of the rectum from the perineum. If the distance is more than a centimeter, the operative approach is abdominoperineal; if the distance is less than a centimeter, a perineal approach can be considered. It is of utmost importance that a fistula from the rectum to the urinary tract be recognized and repaired at the first operation: the postoperative scarring may prevent successful repair at a subsequent procedure.

The postoperative care of these patients includes daily anal dilatation beginning ten days or two weeks after operation. An enterocutaneous anastomosis is always subject to stricture, particularly if the opening is a small one and if the stools are too soft to perform the function of dilatation.

Malrotation

Malrotation is another anomaly that has its origin in the second month of fetal life, when the rapidly growing mid-gut herniates into the base of the umbilical cord. As soon as the abdomen becomes large enough, the gut returns to the abdomen in the following manner: The duodenum slips in posterior and inferior to the mid-gut mesentery, loses its own mesentery and becomes fixed retroperitoneally; after the jejunoileum returns to the abdomen, the ascending colon rotates anterior to the mid-gut mesentery and becomes fixed in the right gutter. The normal posterior attachment of the duodenum and ascending colon provides a long oblique line of fixation of the small bowel mesentery and prevents volvulus of this portion of the intestine. If the duodenum and ascending colon fail to rotate and become fixed posteriorly, the entire mid-gut, from duo-

denum to mid-transverse colon, hangs free on a long mesentery with a single point of fixation—the origin of the superior mesenteric vessels—and is likely to twist into a volvulus.

The symptoms of mid-gut volvulus may occur at any age. Actually, if the bowel is free enough to twist in this fashion, it usually does so in the first few days or weeks of postnatal life, producing the classical signs of intestinal obstruction. Extensive preoperative investigation is unnecessary and may be harmful; the diagnosis of obstruction of unknown etiology is sufficient indication for exploration.

Another form of intestinal obstruction due to malrotation may result from duodenal bands. When the cecum only partially rotates and comes to lie in the epigastrium or right upper quadrant, bands of adhesions from the cecum across the duodenum to the posterior abdominal wall may cause duodenal obstruction. This is usually a partial obstruction which causes variable amounts of vomiting starting soon after birth. In the case of an incomplete obstruction, preoperative gastrointestinal series with contrast material are permissible, and barium enema will demonstrate the abnormal position of the colon.

The operative procedure for malrotation is essentially the same for both volvulus and duodenal bands. The volvulus is reduced and the duodenum freed of adhesions, then the entire colon is placed on the left side of the abdomen. It is advisable to perform appendectomy when possible to avoid delay in diagnosis of left-sided appendicitis in later years.

Omphalocele

As stated above, the mid-gut herniates into the base of the umbilical cord in the second month of fetal life. If this condition persists until birth, omphalocele results. The fact that congenital anomalies are often multiple should always be remembered when treating an omphalocele; if the intestine fails to return to the abdomen, malrotation will result. In patients with omphalocele there is a 30 per cent incidence of malrotation, and intestinal atresia is not uncommon. Routine exploration of the abdomen is not always advised for these patients, particularly if the omphalocele is a large one, but preoperative x-rays which reveal abnormally dilated loops of bowel are certainly indication for exploration.

Anomalies of the Omphalomesenteric Duct

There are several interesting anomalies that result from failure of complete obliteration of the omphalomesenteric duct, the most common being the Meckel's diverticulum, which may bleed, perforate, or cause intussusception. Patency of the entire duct results in drainage of ileal contents from the umbilicus or prolapse of the ileum through the umbilicus. If only a portion of the duct fails to become obliterated, an omphalomesenteric duct cyst will develop. Failure of disintegration of the obliterated duct leaves a cord from the umbilicus to

the ileum, and intestinal obstruction will result from volvulus around the cord. Each of these anomalies should be treated surgically as soon as they are diagnosed.

Duplication

Duplications of the gut are probably formed when the intestine goes through the solid phase of development. If the vacuolization progresses abnormally, one or more vacuoles may not be connected to the lumen of the gut. The duplication may take the form of a cyst or a long tube with one or both ends connected to the lumen of the intestine; hemorrhage, perforation, and intestinal obstruction are the usual complications. The procedure of choice, excision of the duplication, usually involves resection of the adjacent gut, because of the common muscular wall between the two and because the blood supply of the intestine courses directly across the duplication.

Hirschsprung's Disease

The classical case of Hirschsprung's disease is easily diagnosed, but it should be emphasized that this condition may cause death in the newborn period if it is not recognized and treated promptly. The segment most commonly involved is the distal colon, and the defect of autonomic innervation results in the absence of propulsive peristalsis. The feces of an infant are so soft that they will usually pass through this segment with little difficulty, and constipation becomes a problem when the child begins to eat solid food and the feces become firm. Occasionally, however, Hirschsprung's disease will produce intestinal obstruction in an infant a few days or weeks old. X-rays will show a colon obstruction, but barium enema often fails to reveal the etiology because the bowel proximal to the aganglionic segment may not yet be dilated and hypertrophied. If this condition is suspected, the diagnosis may be made by rectal biopsy above the internal sphincter.

Colostomy is usually recommended for the infant with Hirschsprung's disease because of the danger of the severe and sometimes fatal gastroenteritis to which these patients are susceptible. If, however, the colon can be kept deflated by stool softeners and daily enemas, and if the infant exhibits a normal weight gain, conservative treatment may be tried.

The operative procedure of choice consists of resection of the aganglionic segment down to within a centimeter or so of the anus, and end-to-end anastomosis by the pull-through technic. This anastomosis of bowel-to-bowel rarely forms a stricture, in contrast to the enterocutaneous anastomosis for imperforate anus.

If an adequate resection is done, the results of surgery are gratifying and the mortality low. Complications are uncommon, with one exception. These

patients are susceptible to gastroenteritis both before and after operation, with the colon severely distended with gas and liquid feces which cannot be expelled. Vigorous decompression and irrigation through a large rectal tube will usually control the symptoms, and the patient will eventually outgrow the condition.

Meconium Ileus

Meconium ileus is often associated with mucovicidosis. In the absence of the normal pancreatic enzymes the meconium is unusually viscid and tenacious and cannot be propelled through the intestine. The impacted meconium in the terminal ileum causes a mechanical intestinal obstruction shortly after birth, with vomiting, distention, and absence of meconium stools. X-ray will show multiple loops of distended small bowel, usually without fluid levels, and the bubbly appearance of gas mixed in the abnormal meconium. The type of obstruction need not be determined before operation. At laparotomy it is necessary to provide some type of ileostomy or to remove the abnormal meconium—either by lavage or by resection of the impacted segment. Pancreatic enzymes and caroid by mouth and by ileostomy will aid in the removal of the abnormal meconium.

Postoperatively these infants are prone to develop atelectasis because of the abnormal secretions in the respiratory passages, and of course, should they survive the neonatal period, nutritional disturbances will be common. If the patient has mucovicidosis, the prognosis is poor.

Meconium Peritonitis

Meconium peritonitis will develop after rupture of the intestine during intrauterine life. The perforation may be proximal to a congenital obstruction or may occur in a normal intestine. The escaping meconium causes a severe chemical peritonitis with exudation of a large amount of peritoneal fluid. Meconium peritonitis should be suspected in any infant born with a distended abdomen and can be diagnosed by x-rays revealing scattered intraperitoneal calcification. If the perforation has not sealed, massive pneumoperitoneum will develop shortly after birth, and a bacterial infection will be superimposed on the chemical peritonitis. Intestinal obstruction may be due to a congenital lesion or to the many dense adhesions resulting from the peritonitis.

Surgical treatment consists of relieving the obstruction and closing the perforation if it has not healed. The mortality of this condition is high.

Summary

Congenital anomalies of the gastrointestinal tract most commonly result in intestinal obstruction, usually in the neonatal period. Excessive salivation and intermittent cyanosis should always be diagnosed as atresia of the esophagus

until proven otherwise, and the vomiting of bile should be considered indication for immediate investigation.

The use of gastrointestinal series in complete obstruction should be condemned—it only delays operative treatment and permits an opportunity for the aspiration of vomitus.

Until the complications of prematurity and multiple anomalies can be prevented, reduction in mortality from congenital intestinal obstruction will result from early diagnosis and treatment.

Discussion

Dr. O. H. Wangensteen:—You will agree, I am certain, that we have had from Dr. Spencer a very delightful and instructive dissertation upon an intricate and complicated subject.

It was in the hands of N. Logan Leven of my Staff that the first case of esophageal atresia was successfully treated. Shortly thereafter, Dr. William Ladd of Boston did a second case, employing very similar lines of management. Both Leven and Ladd exteriorized the cervical esophagus. In more recent years, owing to the work of Haight, at Ann Arbor, the conventional operation today is to effect a primary anastomosis, if at all feasible.

It would be interesting to know whether Dr. Spencer and pediatric or thoracic surgeons generally who deal with this disorder ever perform exteriorization of the cervical esophagus as Leven did it initially for esophageal atresia. Before Cameron Haight's success with direct anastomosis, Leven, at the University of Minnesota Hospitals had succeeded with the exteriorization operation in a large number of children, who, years later, needed to have their fistulous openings closed and alimentary continuity re-established by bringing up jejunal loops for the purpose. Leven and Varco [*J. Thoracic Surg.* **25**:16-25 (Jan.), 1953] have reported this interesting and unique experience.

I should like to ask Dr. Spencer, when establishing primary continuity, can you always do it by mobilizing tissues locally? Is the defect so wide occasionally that you find it necessary to pull the stomach up, or to employ other devices to re-establish continuity, as we find it necessary to do when operating for cancer of the esophagus in adults?

It was the work of Ladd and Gross at the Children's Hospital in Boston that has made possible present day surgery of duodenal and ileal atresia. When Gross (1953) published his important monograph on pediatric surgery, he indicated that the surgical mortality of correction of this defect was 30 per cent; in other words, survivors numbered 70 per cent. At that time there were perhaps not more than 30 survivors in the entire surgical literature of the world. It is easy to follow, when someone has blazed the trail.

As Dr. Spencer has indicated, in the presence of a pancreatic ring, encircling the duodenum, it is safer not to try to excise the stricturing process but to correct the obstruction by establishing a gastrojejunostomy or duodenojejunostomy.

Strangulation obstructions continue to be one of the important causes of death, in the bowel obstruction problem in both children and adults. We need to effect better means of recognizing this condition when the bowel is still viable. In our clinic, as some of you will remember, Perry [*Surgery* 39:725-735 (May), 1956] suggested inflation of air into the peritoneal cavity to recognize venous strangulations. The sharp difference in density helped to recognize the presence of the infarcted bowel. This agency is of no value in the recognition of arterial strangulations, in that no venous engorgement of the bowel wall occurs.

Bernstein [*Surgery* 44:529-535 (Sept.), 1958] of our clinic finds that he can get aortograms by making an intravenous injection of hypaque, by compressing the extremities at the instant the injection is made. This technic may prove valuable in the detection of obstructions of the superior mesenteric artery. We are sorely in need of objective methods of reorganizing thrombosis of the superior mesenteric artery prior to operation. Such an accomplishment would be a very important forward step in the management of arterial strangulations of the small intestine.

Most intestinal strangulations are essentially venous in nature. And of cases of mesenteric thrombosis perhaps 90 per cent are venous in character. Placement of air in the peritoneal cavity affords the contrast that serves to identify them.

There is one other item upon which I should like to comment, in Dr. Spencer's nice presentation and that is the matter of imperforate anus. I do not know whether she ever employs a little device used in our clinic long years ago [*Ann. Surg.* 92:77-81 (July), 1930] of placing a nickel on the anal plate and taking an x-ray film with the infant in the inverted position. In about 14 hours after birth, gas finds its way normally to the anus. This device serves to inform the surgeon how extensive the imperforation may be.

When I was doing these operations many years ago, I employed the Kraske approach, being encouraged and informed by the roentgen films at what depth I might reasonably expect to find the defect. Today, I believe, many surgeons favor the abdominoperineal approach. On a recent trip to Australia, I had the good fortune of meeting Douglas Stephens who also is sympathetic to use of the perineal approach in correcting imperforation of the rectum. When I prepared the third edition of my book on intestinal obstructions (1955), I recalled for purposes of observation the patients I had operated upon as many as 25 years earlier for imperforate anus. It was a very pleasant surprise to note the number and to find how functional their sphincter control was. All this has been documented elsewhere (*Intestinal Obstructions*, 1955, pp. 439-441).

One of my teachers, Dr. Harry P. Ritchie, was the first to perform the abdominoperineal operation for imperforate anus, a circumstance which I related in the first edition of my book on Bowel Obstructions (1937).

Dr. I. Snapper:—I would like to ask Dr. Spencer a few questions. Do you think that in Hirschsprung's disease the absence of the propulsive waves is, at least, partly due to absence of neurogenic relaxation of the intestine?

I would also like to inquire why reduplication of the duodenum and the upper part of the jejunum is so much rarer than reduplication of the lower part of the small intestine?

Finally, why does spontaneous rupture nearly always occur in the lower left part of the esophagus—2 cm. from the cardia? Is there a special embryological or ontological reason why this part is especially weak?

Dr. Wangensteen:—May I say a word here? I have sat beside my learned friend, Isidore Snapper, on these panels now for several years, and I want to indicate to you how hard it is to accomplish effective missionary work. Through noting the great sensitivity of esophageal epithelium to injury by gastric juice, I was converted long years ago to the belief that spontaneous perforation of the esophagus is acid-peptic perforation of the esophagus. But right here in our own midst, there is a nonbeliever, whose lasting conversion in this important matter, I felt reasonably certain, had been achieved in the first immersion.

Dr. Rowena Spencer:—I would like to thank Dr. Wangensteen and Dr. Snapper for discussing my paper, and I will try to answer the questions.

We do not do only a cervical esophagostomy for tracheoesophageal fistula because of the danger of aspiration of stomach contents. In the presence of a fistula, vomitus goes directly into the trachea. In the absence of a distal esophagus (and therefore absence of a tracheoesophageal fistula) it is safe to do a cervical esophagostomy and feed the infant by gastrostomy.

It is not advisable to bring the entire stomach into the chest of a newborn. Infants take food in a very dilute form, and when they drink a large bottle of milk, the dilated intrathoracic stomach compresses the lung and interferes with respiration. It is permissible to bring a part of the stomach into the chest in order to accomplish a direct anastomosis of the esophagus, but cervical esophagostomy and gastrostomy are preferable to bringing the entire stomach into the chest. When the child reaches the age of a year or so, colon or jejunum can be brought into the mediastinum to bridge the gap between cervical esophagus and stomach.

Yes, we do use the inverted x-rays in imperforate anus to estimate the distance of the rectal pouch from the perineum. We generally don't use a nickel, though; we can't afford it. The little "zero" the radiologist uses to mark the

number on the film is smaller than a nickel and can be fitted right over the little dimple at the site of the external anal sphincter.

I think more surgeons are using the abdominoperineal approach for imperforate anus. I prefer it because the exposure is better and it is easier to find the fistula. With the perineal approach, the incision must be a small one; the tissue planes are not as clearly defined, and a little bleeding obscures the field.

I agree with Dr. Snapper that the absence of relaxation is very important in Hirschsprung's disease. The distal aganglionic segment does not have propulsive peristalsis, and when the bolus of feces reaches this area, the entire segment contracts as a unit. In addition, the absence of relaxation ahead of the wave of peristalsis prevents the feces from entering the abnormal segment. I wonder if there is not some other defect in the remainder of the colon, because of the peculiar gastroenteritis these patients have. It may occur at any time—we have seen it develop a year and a half after surgery, with the child quite ill and severely distended but unable to expel either gas or liquid feces.

I do not know why reduplication of the duodenum and jejunum is not very common, nor do I know about the rupture of the esophagus.

BENIGN POLYPS OF THE DUODENUM*

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and

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Our interest in the subject of benign duodenal polyps was brought about by a recent case of an 11-year old boy who was admitted to Touro Infirmary with a history of abdominal pain and diarrhea of eight years' duration. This boy had been studied by numerous medical centers and on one occasion a diagnosis of tapeworm infestation had been made. He was treated for this condition without improvement and had never responded to any type of treatment.

At Touro Infirmary he was admitted on three occasions and studied completely for the causes of diarrhea and seen in consultation by Dr. Richard Vieth, a pediatrician of our staff. We were all fairly well convinced that this was a functional diarrhea, but due consideration was given to the x-ray findings of a constant filling defect of the first portion of the duodenum, which was thought to be a polyp. On the second admission an exploratory laparotomy was performed, the duodenum was explored, but no polyp was found.

There was a defect in the duodenum produced by an abnormally shaped head of the pancreas. This defect made closure of the duodenum difficult. The conventional transverse closure of the abdominal viscus was impossible and vertical closure seemed to produce a narrowing of the duodenum. He was subsequently readmitted with a stricture of the duodenum that subsided on medical therapy. At the present time he is eating a full diet and still having diarrhea.

With this stimulus we reviewed the records of duodenal polyps at Touro Infirmary for a ten-year period. (January, 1947-January, 1958). In this ten-year period this diagnosis was made on ten patients. The rarity of the disease is well established by this figure, as approximately 194,649 patients were admitted to this institution in this same period.

The earliest definite reference to nonmalignant tumors of the duodenum was made by Rokitansky in his *Lehrbuch der Pathologischen Anatomie*, published in 1861. Cruveilhier in 1835, was the first to report a case of adenoma of the duodenum. Salvioli, in 1876, reported the first adenoma of the duodenum arising from Brunner's glands. Since that time cases of duodenal polyps of mu-

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cosal cell origin have been reported by Perry in 1893, Funkensteen in 1904, Doering in 1907, Wechselmann[1] in 1910, and Willis and Lasersohn[2] in 1925. E. L. King[3] in 1917 reviewed the literature, finding 50 cases of benign tumors of the small intestine. He added 61 cases from Charity Hospital series, bringing the total up to 119 cases. Of these, five were of duodenal origin.

In a review by Hudson and Ingram[4] in 1952 a total of 64 cases of polyps of Brunner's gland origin were reported up to that time. Those authors added one case of their own. Cattell and Pyrtek[5] reported a polyp in the third portion of the duodenum in 1949. Edwards and McHardy[6], in 1950, reported a polyp in the first portion of the duodenum. In 1951 an adenoma of the second portion of the duodenum was reported by Noon[7]. Culver and Caccese[8] reported a case of duodenal polyp as recently as 1957.

There are certain definite characteristics of the small intestine which differentiate it from the stomach and colon. Embryologically the small intestine is a

TABLE I

Age	No.
20-29	1
30-39	0
40-49	1
50-59	1
60-69	5
70-79	1
80-89	1
Total	10

smooth epithelial lined tube that had undergone rapid elongation in embryo during the latter four months of fetal life. The small intestine is a coiled tube of relatively uniform size, the lining of which is characterized by villi between which lie crypts of Liebarkuhn. The mucous glands of Brunner are found in the submucosa of the duodenum and upper jejunum while the terminal ileum harbors collections of lymphoid tissue known as Peyer's patches. The food passes rapidly through the gut with little or no stasis. Absorption is greater in the small intestine than in any other part of the gastrointestinal tract[9].

Saint[10] points out that the glandular tissue at some point in the mucosa hypertrophies and becomes hyperplastic. This makes an apparent fold in the mucosa. Shortly thereafter the submucosa projects slightly upward, which is the beginning of the pedicle. The glandular tissue over the polyp increases in amount and is continuous with the normal mucosa. The development of the pedicle occurs because of drag upon the tumor by a constant flow of material over it and because of the peristaltic contractions of the bowel endeavoring to expel the foreign body.

Two hypotheses have been forwarded as to the cause of adenomata: 1. Inflammatory and 2. Primary epithelial change. There is strong evidence of development of adenomata in chronic inflammatory conditions as pointed out by Hudson and Ingram[4]. On the other hand, Hauser and Bardenheuer, as quoted by Saint[10], feel that the inflammatory reaction is secondary to the epithelial change.

Raiford[9], in his publication of 1932, points out that the glandular elements of a typical adenoma show two types of cellular development. Near the pedicle the cells are normal in appearance, having dark oval nuclei lying at the base of columnar cells distended with mucus. The cells become more pleomorphic, with increased nuclei, granular cytoplasm, and loss of polarity as they reach the periphery. The pedicle is composed of connective tissue arising from the submucoa and ramifying between the glandular elements of the tumor. Grossly, the mucosa

TABLE II

SYMPTOMS

Upper abdominal pain	3
Gastrointestinal bleeding	3
Nausea and vomiting	2
Fever	2
Anorexia	2
Weight loss	2
Diarrhea	2
Flatulence	1
Weakness	1
Jaundice	1
Total	17

is intact and red. On cross section it has a mushroom-like appearance with a central white fibrous stalk arising from the gastrointestinal wall.

In our review of cases at Touro Infirmary we found that the age limits ranged from 29 through 82 years.

The age distribution of this group shows that 50 per cent of the polyps occurred between the ages of 60-69 and 90 per cent occurred after the age of 40. This would indicate that benign duodenal polyps are uncommon in children. Polyps in other parts of the gastrointestinal tract are also noted in this older age group. Weishaupt in 1916, as reported by Golden[1], recorded the case of an 11-day old infant with a Brunner's gland polyp in the duodenum. Raiford[9] in 1932 in a review of tumors of the small intestine reported a proven duodenal polyp in a child of six months. With improved roentgenological technic in the past 20 years the preoperative diagnosis of duodenal polyp has become more common.

This has been shown by the publications of Ochsner and Kleckner[11], Culver and Caccese[8], and Hudson and Ingram[4].

In our cases at Touro Infirmary we could not determine a prediagnostic syndrome compatible with the diagnosis of duodenal polyp. The common symptoms found in these patients are shown in Table II.

From this table the symptoms are seen to be common to many gastrointestinal diseases. Hudson and Ingram[4] stated that "80 per cent of cases have associated epigastric distress with fullness and eructation. However, symptoms are not characteristic. The larger tumors may even go on to obstruction. With ulceration of the mucosal lining of the tumor, hemorrhage may occur." Cattell and Pyrtek[5] point out that melena may be the presenting symptom, the result of ulceration. In the absence of gastric or duodenal ulcer one must consider duodenal polyp as a possible cause of gastrointestinal hemorrhage. Culver and Caccese[8] stress the fact that duodenal polyp may simulate any disease of the gastrointestinal tract.

TABLE III

SURGICAL TREATMENT

Duodenotomy with excision of polyp	2
Gastrotomy with excision of polyp	1
Subtotal gastric resection and excision of polyp	1
Cholecystoduodenostomy with biopsy of polyp	1
Total	5

The treatment of these benign lesions, if thought to be symptomatic, is duodenotomy and excision of the polyp. In reviewing our records at Touro Infirmary we found that the surgeons operated upon five patients, an incidence of 50 per cent of the cases. One patient admitted to the Surgical Service was in such poor condition from associated renal disease that surgical excision of the polyp was not considered. The four remaining patients were admitted to the Medical Service without benefit of surgical consultation.

There was considerable difference in the surgical aproach to this lesion in these five patients as noted in Table III.

There was no operative mortality from the surgical treatment of these polyps. In one case treated by surgery a by-pass procedure was performed on an 82-year old patient who was thought to have an adenocarcinoma of the ampulla of Vater. Tissue study of the biopsied lesion later revealed the benign character of the polypoid mass. The patient having a subtotal gastric resection was brought to surgery because of severe weight loss, and the operator felt that he was dealing with a malignant lesion of the stomach in addition to the duodenal polyp. From perusal of the literature there is no uniform method of sur-

gical therapy. Earlier authors have reported various resectional and by-pass procedures in the treatment of this lesion[12]. We feel, however, that from a technical and anatomical standpoint there would be some hazard associated with any of the above mentioned procedures. We are of the opinion that duodenotomy with excision of the polyp is adequate treatment.

The type of tumor found on these surgical specimens were as shown in Table IV.

TABLE IV

From Brunner's glands	3
From mucosal cells	2

Hoffman and Grayzel[13], in their publication of 1945, point out that 10.6 per cent of all benign tumors of the duodenum arose from Brunner's glands and 31.8 per cent arose from mucosal cells. It was of interest to us that in this same series 12.1 per cent of the defects of the duodenum were produced by some type of pancreatic tissue.

Summary

In summary, ten patients were found at Touro Infirmary to have benign duodenal polyps during the ten-year period between January, 1947 to January, 1958. A brief review of embryology and histology was given. Because of lack of stasis and the alkalinity of the duodenum tumors may be less common in this region. We know that tumors, both benign and malignant increase in frequency as the terminal ileum is approached. The rapid elongation of the small intestine late in fetal life may be a factor in possible freedom from fetal rests and rests of development. In our series the age of incidence ranged from 20 to 90 years, with 90 per cent occurring after the age of 40 years. We could not find any definite symptom-complex characteristic of this lesion. When symptomatic, it may simulate any gastric, duodenal or biliopancreatic lesion. In the absence of any demonstrable cause of hemorrhage, benign duodenal polyp should be suspected. The diagnosis must be kept in mind, but can be confirmed by roentgenological studies. The final diagnosis rests, of course, upon microscopic inspection. The treatment of choice is duodenotomy with excision of the polyp.

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Discussion

Dr. O. H. Wangensteen:—Obviously, this is a rare lesion. I would like to ask Dr. McQuitty whether he believes that the duodenal polyp, like polyps elsewhere, especially in the colon and rectum, are precursors of cancer. I am inclined to think so, and I would include gastric polyps too—the latter being rather slow precursors of gastric cancer. We are completely dependent, of course, upon the roentgenologist in the recognition of such rare and obscure lesions as duodenal polyps.

The three cases I have seen have all been treated by doudenotomy. There is one lesion which has occasioned us some difficulty in differential diagnosis. I should like to ask Dr. McQuitty whether he has had any trouble with a prolapsing bit of antral mucosa in the differential diagnoses. In my experience, this latter condition causes a good deal of pain whereas duodenal polyps are heralded usually only by hemorrhage.

Some of you will remember that Dr. McKittrick presented a case of duodenal cancer at the Clinicopathological Conference last year. The persistent defect in the film helped establish the diagnosis in that instance. Is its antecedent a polyp which you or I may be slow to diagnose? I think it is.

Dr. I. Snapper:—A duodenal cancer is extremely rare, and the few cases I have seen were ulcerating and not polypoid in character. I, therefore, doubt whether a benign polyp of the duodenum is a precancerous disease. If, however, the polyp is ulcerated and bleeds then, of course, there is decidedly an indication for operation.

In my opinion one should avoid the term "polyp" as much as possible. These so-called polyps of the duodenum are truly adenomas as Dr. McQuitty correctly called them. Let us avoid the confusion which exists in the pathology of the colon. Here, mucosal tags which are the result of an inflammation of the colon

are called polyps, whereas congenital adenomatosis is also designated as polyposis. Since congenital adenomatosis or polyposis often causes carcinoma of the colon, the "polyps" in ulcerative colitis which are only small mucosal tags, are also considered to be potentially malignant. It cannot be denied that the roentgenologic picture of the late stages of ulcerative colitis may be very similar to that of a congenital polyposis. This is another reason why so many surgeons feel that the mucosal tags or "polyps" are allegedly the reason why in ulcerative colitis carcinoma of the colon occurs more frequently than in the rest of the population.

Nevertheless, many surgeons do not incriminate the innocent mucosal tags anymore and explain the increased cancer incidence in ulcerative colitis differently. They reason that a preexisting adenoma of the colon will develop malignant degeneration much more rapidly under influence of the irritation caused by an ulcerative colitis than if the adenoma was present in a noninflamed colon.

The lesion Dr. McQuitty described is a true adenoma, and we should avoid the word "polyp" in order to keep our semantics clear and avoid confusion as has developed about the cancerogenic tendency of the mucosal tags in ulcerative colitis.

Dr. James T. McQuitty:—I certainly appreciate the comments of these discussers. I feel that this sort of comment on a subject of such a rare disease represents the thought of two men who have given a great deal of time to diseases and to thinking about pathology.

The terminology is often confusing, Dr. Snapper, and I agree with you that you have an excellent point. I cannot help believing, though, and I personally trust the polyp about as much as I do a carcinoma. I feel that they are all premalignant lesions.

We attempted to find a correlation between duodenal cancer and polyps but we didn't succeed in doing so. I don't see how we can get away from the fact that the higher the incidence of carcinoma of the gastrointestinal tract, the higher the incidence of polyp formation in those regions.

Dr. Snapper:—Adenomas!

Dr. McQuitty:—All right. After the adenomas become malignant, they ulcerate, so, from the fact that most of the ulcerating carcinomas are in the duodenum, I feel that an ulcerating adenoma can be found to give rise to a to approximately 8 mm. over a distance of 10 cm. It is, thus, about the size carcinoma just as well as a carcinoma can arise from an ulcerating lesion.

THE CLINICAL EVALUATION OF OXYPHENCYCLIMINE IN PATIENTS WITH PEPTIC ULCER

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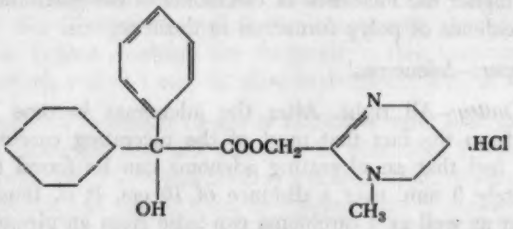
Most clinicians and physiologists consider the role of gastric acid secretion highly significant in the pathogenesis of duodenal ulcer. It is therefore not surprising to find that antacid and antisecretory agents in general have been widely studied in the therapy of this disorder. Among the most intensively investigated have been those drugs that block the effect of the vagus nerves on gastric secretion.

A recent and by no means complete review of these anticholinergic compounds[1] lists 305 references. Fourteen such synthetic compounds are included in this review and many others exist. Despite the multiplicity of such compounds and the apparent effectiveness of many of them, there seems to be room for improvement. One of the obvious shortcomings of most available anticholinergics is the relatively short duration of action. This of course is of particular importance at night, when the relatively high basal or interdigestive gastric secretion of ulcer patients is essentially unopposed by agents whose action is of short duration, and patients find it inconvenient and upsetting to awaken for purposes of repeated medication. In recognition of this serious problem we have long advocated the use of continuous intragastric drip therapy[2]. The continuous drip is intended primarily for management of the ulcer patient with acute and severe symptoms. As, however, an adjunct to this therapy in the severe cases, for the initial treatment in the milder cases, and, for day-to-day maintenance of all patients, we have need for a potent anticholinergic drug with a prolonged action.

One such agent was recently brought to our attention. This substance, oxyphencyclimine hydrochloride*, has the following structural formula:



It is unique in its tetrahydro-pyrimidal ring, which differentiates it from the quaternary amine structure usually employed among anticholinergic drugs.

*Available from Pfizer Laboratories as Daricon. Materials for this study supplied by the Clinical Research Dept., Chas. Pfizer & Co., Inc., Brooklyn, N. Y.

In rats and dogs it has been found[3] to be an effective anticholinergic, antisecretory agent, without curare-like activity and with little or no ganglionic-blocking action. It was shown to be the most effective of a series of known antisecretory agents in inhibiting gastric secretion in the 5-hour Shay rat and in preventing rumenal ulcer formation in the 17-hour Shay rat. Spontaneous, histamine and meal-induced gastric secretions in chronic gastric fistula dogs, were inhibited for more than three hours as to volume and acid content by single doses of 0.05 mg./kg. of oxyphencyclimine intramuscularly and 0.1 mg./kg. orally. A relatively slight degree of depression of salivary flow was found and tachycardia was not noted. Preliminary studies in human subjects[4] showed evidence of potency and a prolonged duration of action.

With this background it seemed reasonable to proceed with an investigation of the antisecretory effects of oxyphencyclimine in patients with peptic ulcer.

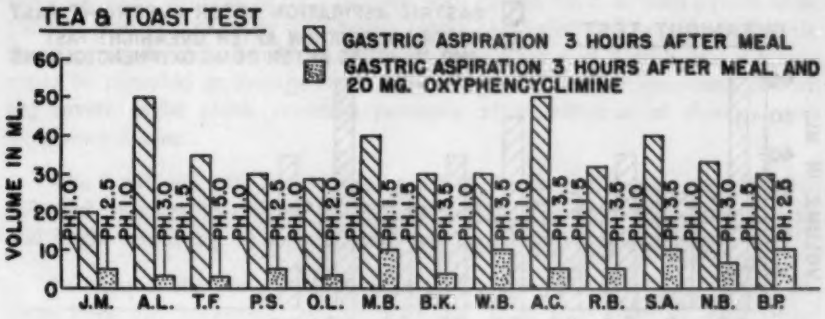


Fig. 1

Objective Studies

To assess the antisecretory activity of oxyphencyclimine, the effects on food-stimulated gastric secretions were measured. For purposes of this test each patient was given 20 mg. oxyphencyclimine after an overnight fast. Fifteen minutes later the patient was given a breakfast consisting of 120 c.c. of weak tea and two slices of dry white toast. Three hours afterward the stomach was aspirated as completely as possible. Aspirates were measured for volume, pH was determined with the use of Hydrion indicator paper, and the character of secretion was noted. As a control, each patient was retested on a different day, following the same procedure outlined above, but without administration of the drug.

Thirteen patients with known duodenal ulcers were investigated in this semiquantitative manner. Results are depicted in Figure 1. In all instances there was a marked decrease in volume of gastric juice 3 hours after the drug. In

most cases there was a noticeable difference in the character of the material aspirated; control specimens were more watery than the thick, mucoid samples obtained after the drug. Definite favorable effects in pH were also observed. In 12 of the 13 cases there were significant increases (pH change of 1.0 or more). In 8 patients a pH of 3.0 or higher was achieved by the use of oxyphencyclimine.

In order to evaluate the alleged prolonged activity of oxyphencyclimine, an overnight test was performed. A 20 mg. dose was given at approximately 10 P.M. in the evening before aspiration. After an overnight fast, gastric secretions were aspirated starting at 9 A.M. (11 hours after drug dose). For control purposes each patient was re-examined on a different day after an overnight fast without drug. Specimens were measured for volume, pH was determined with Hydrion indicator paper, and the character of secretion was noted.

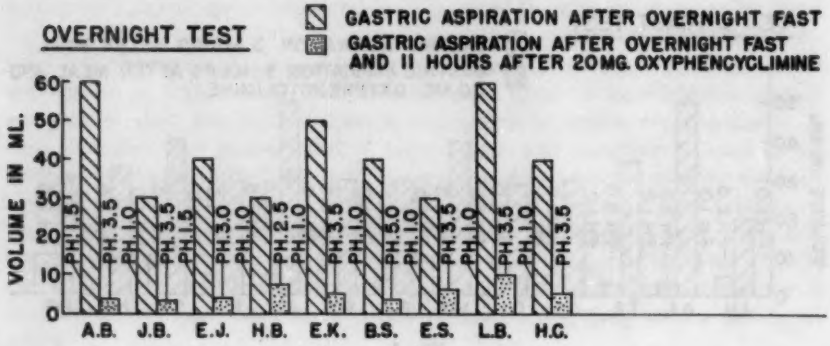


Fig. 2

Nine duodenal ulcer patients were studied with the overnight test. The findings are shown in Figure 2. In all instances there was a marked diminution in gastric juice 11 hours after the drug. The postdrug secretions were also more mucoid and less watery than those samples obtained during the control test. Significant increases in pH were seen in all patients, and in 8 of the 9 cases investigated a pH of 3.0 or higher was obtained with oxyphencyclimine.

Clinical Studies

During the past year a total of 96 patients has been treated with oxyphencyclimine for symptomatic relief. Prior to therapy the specific diagnosis in every case was established by history and physical examination, x-ray, and any other indicated procedure. Many of these patients had previously demonstrated refractoriness to numerous other drugs. All patients in this study were on a liberal ulcer diet, but did not receive any adjunctive therapy.

The study series consisted of 80 patients with duodenal ulcer, 7 with gastric ulcer, 2 with jejunal ulcer, and 7 with hiatus hernia and peptic esophagitis. Seven of these patients have received the drug and have been followed continuously for one year, 18 patients have been treated for periods of two to six months, 61 have been treated for one to two months and 10 were treated for periods of two to four weeks.

The usual dose of oxyphencyclimine was 10 mg. given twice daily, usually before breakfast and upon retiring. Six patients with severe night pains had their evening doses increased to 20 mg. After the acute phase of their illness, 24 patients were maintained on a single daily dose of 10 mg.; 17 of these cases have now been controlled nicely for four months or more on the once a day schedule.

Good symptomatic responses were achieved and maintained in 77 of the duodenal ulcer patients, in all 7 patients with gastric ulcer, in both jejunal ulcer patients, and in 5 of those with hiatus hernia and associated peptic esophagitis. In other words, only 3 patients with duodenal ulcer and 2 with hiatus hernia could be regarded as therapeutic failures. Response of ulcer symptoms, including severe night pains, occurred promptly after initiation of therapy with oxyphencyclimine.

The 7 patients with gastric ulcer and 5 of the duodenal ulcer patients were followed radiographically. In one patient healing occurred within two weeks, and in the remaining 11 cases healing was complete within a month.

In none of the patients on continuous therapy with oxyphencyclimine has there been any recurrence or complications. It is recognized of course that longer periods of observation will be required before a significant decrease in frequency of complications can be specifically attributed to the use of this drug.

Side-effects were sufficiently objectionable in 4 patients to require discontinuation of therapy. These patients usually had one or several of the following complaints—moderately severe dryness of the mouth, blurring of vision, constipation, or urinary hesitancy. Virtually all patients in the study had some degree of dryness of the mouth. This, however, was generally quite mild, and often diminished or disappeared with continued therapy.

Summary and Conclusions

Oxyphencyclimine is a potent, long-acting anticholinergic agent. Secretory inhibition was apparent three hours after 20 mg. in a series of 13 ulcer patients, in 8 of whom a marked inhibitory effect was noted. Similar activity was observed 11 hours after 20 mg. oxyphencyclimine in a series of 9 ulcer patients, in 8 of whom marked inhibitory effects were noted. Good symptomatic responses were seen in 91 of 96 treated for periods up to one year with average doses of

10 mg. twice daily. Objectionable side actions were present in only 4 of the clinical series. Oxyphencyclimine appears to be a valuable agent, useful as an adjunct in the management of acute severe ulcer patients, for the initial treatment in the milder acute cases, and for day-to-day maintenance of all peptic ulcer patients.

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AMEBIC DYSENTERY IN MEXICO*

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Incidence

Amebiasis in Mexico is of special importance because of the large number of people affected with this protozoa. It is found in all of the states in the Republic of Mexico, but it is more prevalent in the tropical zones and especially along the sea coasts of the south and southeast.

In the City of Mexico patients with amebiasis are very numerous, as was proven by Bustos' study, which showed that in a group of 1,000 children 57 per cent were infested with this parasite. Ramírez Ulloa's study showed that among 800 patients who presented symptoms of colitis, 66 per cent had *Endameba histolytica*. Beltrán and Larenas' investigation proved that amebas were found in 47 per cent of 410 samples of feces obtained in an educational center.

In the Division of Gastroenterology of the General Hospital of Mexico City, more than 70 per cent of our patients have amebas in their fecal matter. Some of them have symptoms, but the majority are carriers without symptoms. The high rate of prevalence is due to the fact that only patients who have trouble with their digestive system and who belong to the lowest income group are received at this division of the Hospital.

Classification

Craig's clinical classification is, in our opinion, the most practical. He describes five type of patients with amebas:

Group I—Nonsymptomatic carriers.

Group II—Patients who have indefinite symptoms in their digestive system and in their nervous system and who often are included in the carrier group.

Group III—Those who suffer recurrent attacks of diarrhea, in addition to the symptoms mentioned in group II.

Group IV—Those who suffer from dysentery, either acute or chronic.

Group V—Those who suffer from complications of amebiasis, such as abscess of the liver, etc.

Symptoms

In our practice (hospitals and private) nearly all of the patients whom we have attended fall into the first three groups. In our experience the symptoms

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which are most frequently found among these three groups and which lead us to look for amebas in the feces are: diarrhea alternating with constipation, mucus in the stools, meteorism and vague abdominal pains, indefinite or similar to intestinal pains.

Class IV involving dysentery is not prevalent. It is apparent that this type has diminished in the last few years. This may be due to the generalized use of sulfas and antibiotics. For this reason we shall not limit ourselves to it in this paper. We have chosen to present, in a general but concise form, the problems of amebiasis in our country.

The symptomatology in the cases which we have seen is completely similar to that described in the classical medical textbooks.

Class V has a particular interest for us because of the great frequency with which we see patients with hepatic amebiasis, with or without complications.

Complications

The complications which may be found in the course of intestinal amebiasis are numerous: appendicitis, intestinal perforations, granuloma of the colon, cutaneous lesions, hepatic amebiasis (the most prevalent of all), etc.

We have seen several cases of amebic appendicitis, identified as such by the pathologist, after surgery or at necropsy. We believe that these cases would be found more frequently if amebic lesions and trophozoites of amebas were looked for in all appendices removed.

Intestinal perforations are not frequent in our experience. In 1,016 autopsies performed during the last three years at the General Hospital of Mexico we have only encountered three cases of this nature. Two of them were found in patients of the Gastroenterology Division.

In one case we found amebic colitis, ulcerated and perforated at the level of the appendix, with a retrocecal abscess. When alive, the patient did not present a dysenteric syndrome. In another case we found intestinal amebiasis with ulcers in the cecum, ascending colon, transverse colon and rectum, with perforation of the cecum to the peritoneal cavity and to the retroperitoneal tissues of the right ileac cavity.

The third case presented amebiasis of the colon with five perforations to the abdominal cavity, granuloma of the rectum and hepatic microabscess with trophozoites of *Endameba histolytica*. This patient had dysentery for three months and after a rectal examination a vegetating and bloody tumor was found, with the appearance of carcinoma. A biopsy revealed amebiasis of the rectum. Emetine was given to the patient during several days and a second rectal exami-

nation was made. The tumor was found to have changed. At that time it had the appearance of necrosis rather than of a vegetating tumor.

Granulomata also are rare. In addition to the case mentioned we know two more, both localized in the transverse colon. In both cases the clinical and radiological diagnosis was cancer of the colon, and in both instances a colectomy was performed. The correct diagnosis was made by the pathologist.

Cutaneous lesions produced by ameba are even less frequent. We have had only one proven case of amebiasis of the anal margin.

Hepatic amebiasis is the most frequent of the complications of intestinal amebiasis in Mexico. In the past 15 years we have seen more than 230 cases at the Gastroenterology Division of the General Hospital. The percentage of frequency in relation to all the other diseases seen during this time was 3.7. We shall not analyze these cases since it is outside the scope of this paper, but it is important to note that the mortality rate which we have is low and in the last few years has become lower still perhaps because of the influence of new, therapeutic drugs.

We should like to avail ourselves of this opportunity to show a procedure which was introduced by one of the doctors at the General Hospital and which we have adopted, since it is useful in the management of these patients. We are referring to the injection of air into the cavity of the abscess, once the abscess has been drained through a trocar. This "pneumoabscess" is clearly visible in the x-ray films and by this means its course can be closely followed, since it is easy to see the reduction of the cavity as the process improves.

By using this method at the Gastroenterology Division we are able to obtain the radiographs which I shall proceed to show to you, and which are probably unique. They correspond to a case of abscess of the left lobe of the liver open to the pericardium. The abscess was punctured and evacuated and air was injected through a trocar. In the film, the cavity of the abscess and the pneumopericardium may be seen clearly. This patient is the only one among four cases which we have observed who survived this grave complication.

Treatment

There is little diversity of opinion regarding the treatment of acute amebic dysentery in Mexico. As in other places, however, there is no agreement as to the ideal treatment for intestinal amebiasis in the three first groups of the classification of Craig that is, the nondysenteric forms. We believe that the ideal treatment for these forms does not exist at present, and that we should continue to search for more effective agents.

In the acute forms, we always use emetine hydrochloride in ampules of 4 to 6 centigrams intramuscularly during 4 or 5 days, in combination with sulfas

or antibiotics, especially the tetracyclines. Rest and proper hydration are also necessary. With these measures, good results are obtained in a short time in most cases.

Emetine is the drug which we employ in the complications of intestinal amebiasis. We have had a great deal of experience in using it in hepatic amebiasis since we inject, in the case of adults 6 or 8 centigrams daily up to a maximum of 60 or 70 centigrams (1 centigram per kilo of weight as a maximum dose). This means that several thousand injections of emetine have been given in the Division of Gastroenterology of the General Hospital. We have not seen, up to the present time, any serious accident caused by the use of this drug.

We are aware of the fact that emetine is toxic and that it accumulates in the body, but we have become accustomed to its use and we do not hesitate in using it in our patients. We advise that care be taken, however, in order that any toxic manifestations such as vomiting, diarrhea, pains in the legs, arterial hypotension, etc., be noted, in which case the medication should be immediately suspended.

We systematically keep an electrocardiographic control of these patients. Recently Dr. Cabrera, of the National Heart Institute of Mexico City, indicated that the changes in the T-wave in the electrocardiogram during the use of emetine should not necessarily be interpreted as pathological changes brought about by the use of this drug.

In many cases during the last few years we have used cloroquine, in addition to emetine, in treating hepatic amebiasis. We inject emetine first until we reach the maximum dosage and then we administer diphosphate of cloroquine (Aralen) or sulfate of hydroxycloroquine (Plaquinol) as long as necessary.

At the Hospital of Nutrition of the City of Mexico only cloroquine is used in the treatment of hepatic amebiasis and the results obtained have been satisfactory.

In the nondysenteric forms of intestinal amebiasis, we have used all of the drugs which have been available for this during the past few years: arsenicals, iodoclorooxiquinoleins, diiodohydroxyquinoleins, bismuthglicolilarsenilates, pontalin, camoform; antibiotics: terramycin, fumagilin, stylomycin, etc.

Recently we had the opportunity of using stylomycin in 51 patients who had cysts of *Endameba histolytica* in their stools. In each case we administered the same dose, regardless of the weight and age of the patient (we purposely used a low dosage of 300 mg. daily up to a total of 1,000 mg.) during four days of treatment. In 47 out of 51 cases the cysts disappeared after one treatment. In two cases they disappeared after the second treatment and in only one case they continued to persist in spite of the two treatments.

Dr. Echeverría, of the Military Hospital of Mexico City, treated 28 patients, obtaining similar results in cyst carriers, but he found it necessary to use a combination of stylomycin and achromycin in order to eliminate the trophozoite and rectal ulcerations in the cases in which these conditions were present.

Fumagilin is not well tolerated by our patients and the results, in general terms, have not been very satisfactory. The same may be said of most of the drugs which are employed in an isolated form. For this reason we continue to use the combination of two or more drugs, usually diodoquin and carbarsone and later fumagilin, terramycin or wintodon.

The results obtained in this way are good, even though the treatment is long and troublesome.

AN ADDITIONAL METHOD FOR RECORDING INTUBATION STUDIES OF THE HUMAN INTESTINE

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Denver, Colo.

Intubation studies in the small intestine have been carried out many times in the past. It is still possible to state as Ingelfinger did in 1943[4] that a balloon method free of objectionable features has yet to be devised. It is realized that the balloon itself in the intestine serves as a constant and abnormal source of stimulation.

The purpose of this paper is to describe a newer and possibly more sensitive kymograph recording of pressures from a balloon in the intestine. With this method, a more exact and reproducible picture may be obtained by the recording apparatus. This is true even though the major objections to balloon study have not been met. In addition, this device has initially been used to study the intestinal motility in hypermotility states such as nonspecific diarrhea before and after the use specifically of Donnagel®*. From this study, we believe we have another tool for the study of intestinal motility.

The purpose of this study was to perfect the recording device and explore its possibilities. The drug, Donnagel, was used to demonstrate whether or not it had an effect in slowing the hypermotile bowel as recorded by this device.

Method

The method hereafter described was first used by Mr. C. J. Hlad at the Veterans Hospital, Denver, Colorado, in 1955†. Modifications in his original technic will be described later. The usual balloon method was used after introducing the tube into the stomach and small intestine. The variations in activity of the balloon were transmitted to the radioactive material in the diaphragm arrangement. The activity of the radioactive material, sodium in this case, was then picked up by a scanner and the results were recorded on a revolving kymographic drum (Fig. 1).

The variations used in this technic were made applicable to small intestine studies. A smaller lumen tube was used than was found necessary in the stomach. It was also found important to have a constant pressure of air in the balloon at the end of the tube in the intestine. Of further interest was the fact that the tube must be kept in place in the small intestine for some time before

From the Department of Medicine, General Rose Memorial Hospital, Denver, Colo.

*Donnagel®, an antidiarrheal preparation containing koalin, pectin, dihydroxy aluminum aminoacetate, along with the belladonna alkaloids and phenobarbital as present in the Donnatal formula, was supplied by the A. H. Robins Co., Inc., Richmond, Va.

†Unpublished data.

reproducible kymographic recordings were found. At times it was necessary to wait up to a half-hour for these readings to stabilize.

With these modifications the sensitivity of the instrument was found to be greater than any previous recording instrument in the small intestine that we tried, and not as variable.

The only attempt at a controlled experiment was the use of a prepared substance that looked like Donnagel without any of its active ingredients. This was used on the same patient as the regular Donnagel in four instances and on different patients in six instances.

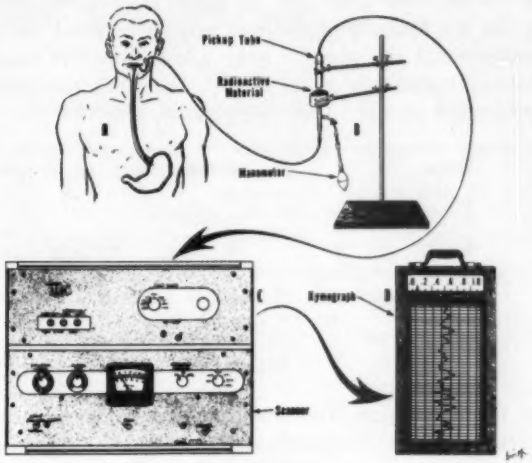

Fig. 1

Subjects

A total of 56 persons and patients served as subjects and a kymographic recording was made from each. Many of the records were taken from the stomach and into the small intestine at different levels. Of these 56 subjects, 30 could be considered normal controls, in that there was no clinical abnormality of intestinal activity noted. Each of the remaining 26 patients had nonspecific diarrhea associated with distinct hypermotility during the time the recordings were made. Recordings were done during the stage of hypermotility and after the use of Donnagel.

Procedure

The tip of the tube under fluoroscopic control was allowed to be in the stomach or in the small intestine. Once in the small intestine, recordings could

be taken at frequent intervals as the tube was carried down. The passage of the tube could be stopped at any point and x-ray verification of its position could be determined. The main interest was in the motility of any portion of the small intestine so that the exact location was not of paramount interest.

Results

The nature of the record obtained can be seen in the accompanying graphs (Fig. 2). These correlate rather well with those previously recorded. All of the patients with diarrhea having clinical evidence of hypermotility showed disordered rhythm as compared with the normals.

From the studies with Donnagel, 20 different patients were used. Six of the 20 were given the Donnagel-appearing mixture without the active ingredients. The Donnagel and the placebo were administered in the same manner, namely one tablespoonful four times a day. No other medication or dietary management was used. Fourteen of the patients received the standard Don-

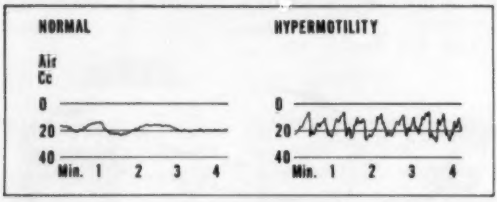

Fig. 2

nagel. Four of the patients received the standard Donnagel after the placebo had been tried for two days. The medication was continued for four days unless the bowel returned to normal earlier clinically and according to kymographic recordings.

The results in these patients showed that the standard Donnagel had an effect after the first day of administration of reducing hypermotility of the bowel as measured by this recording device to a more nearly normal pattern. The Donnagel-type placebo in each of the six instances did not seem to have the effect on the irritability of the bowel.

Comment

The apparent usefulness of kymographic recordings in studies of intestinal motility in the human subject has previously been demonstrated[1,2,3,4,5]. A new technic for recording this arrangement will always be of value and may lead to a further verification of old principles or a modification of them.

Further studies will be done with this modality to determine the reaction in disease states other than hypermotility. The use of an antidiarrheal agent

was chosen to study the efficiency of the recording system as well as the drug. No attempt as yet was made to correlate the efficiency of other known antidiarrheic agents as compared with the one used.

Conclusions

1. On the basis of the 56 kymograph recordings of intestinal activity, a new improved method of recording is presented.

2. Studies made on hypermotile bowel states accompanied by diarrhea revealed graphic changes in the recordings made.

3. The drug, Donnagel, when used, demonstrated a definite slowing effect on the bowel activity after the first day of administration.

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EDITORIAL

"Hypertrophic" Gastritis

The term "hypertrophic" gastritis continues to be employed extensively by gastroscopists, radiologists, internists and even gastroenterologists. This despite numerous reports of the inability to correlate this diagnostic misnomer with pathologic studies. More recently gastroscopists in the face of consistently negative histology have more cautiously referred to this as "visual hypertrophic gastritis". Gitlitz and Colp[1] studied resected gastric specimens following surgery for duodenal ulcer. They classified gastritis into superficial, interstitial and atrophic forms. The entity of hypertrophic gastritis was considered extremely rare and not encountered in their series. More recently, Jaske, Finckh and Wood[2] in their comprehensive monograph (that should be recognized as a minor if not a major classic) utilizing the ingenious and simple Wood biopsy instrument studied 1,000 gastric biopsies. These authors classified gastritis into superficial gastritis and various forms of atrophic gastritis and finally, gastric atrophy. Hypertrophic gastritis is not included in their pathologic classification.

Menetrier[3] described two forms of a so-called hypertrophic gastritis that he believed were precursors of carcinoma, *polyadenome polypeux* and *polyadenome-en-nappe*. The former is characteristic of multiple polypoid gastric tumors or adenomata and the latter has been described as giant rugal folds with beading of the mucosa presumably associated with diffuse hyperplasia. The pathology of adenomata is well known. The second form is a little more complicated. Giant rugal folds are rarely visualized following a barium meal examination. Although 85 cases have been reported up to 1955, undoubtedly considerably more have been seen. Giant rugal folds are usually caused by diffuse hyperplasia of glandular mucosal elements. The differentiation between giant rugal folds and the prominent rugal folds that the gastroscopist and the radiologist often describe as hypertrophic gastritis is more than a matter of degree.

Duval, Roux, Beclere and Moutier[4] found that prominent gastric rugal folds seen roentgenologically bear no correlation to the gastroscopic, surgical or histological appearance. In these folds they found a slight increase in connective tissue surrounding a vascular core but at their bases and at their summits there was pronounced thickening of the *muscularis mucosae*. The alterations in the height and thickness of the gastric mucosa appeared to depend on alterations in the thickness of the *muscularis mucosae*. They, and subsequently others, concluded that prominent rugal folds simply indicate an increase in the tone or irritability of the *muscularis mucosae*. An iritable stomach reveals many more folds than an atonic one. In our own experience we have found little alteration in the character of the mucosal folds during repeated examinations in the same patient; the character of the folds appear to be more or less constant for the individual. As regards the presence of pathology, in general, one can say that

the more prominent the folds (in height rather than width) the less the likelihood of pathology and the thinner or flatter the folds the greater the likelihood of significant mucosal alteration.

The clinical diagnosis of gastritis which occasionally serves to mask other conditions should be deplored unless it has been confirmed by gastric biopsy[5]. Not infrequently surgery has been performed because prominent folds were considered to be lymphosarcoma or more rarely carcinoma. Such surgery can be avoided by employing gastric biopsy to evaluate the nature of an apparently diffuse involvement of the gastric mucosa. The Wood tube is a safe instrument and easily passed. The specimens obtained are adequate and diagnostic. Its use has already served to classify the pathology of inflammatory diseases of the stomach *in vivo* and to initiate greater interest in this subject.

A conscientious effort to avoid the use of the term "hypertrophic" gastritis eventually will clear up some of the clinical confusion surrounding the diagnosis and treatment of nonspecific inflammatory diseases of the stomach.

A. I. FRIEDMAN, M.D., F.A.C.G.

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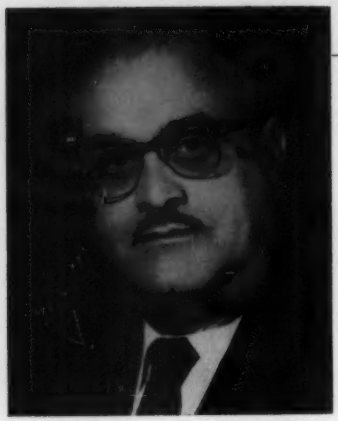

President's Message

Awards

At the present time, the Research Committee of the American College of Gastroenterology is engaged in evaluating entries of unpublished original work in gastroenterology, received in competition for the Henry G. Rudner, Sr. Award.

The contest, which was announced last fall and which we hope to sponsor annually, offers a cash prize plus a travel allowance for the winner to present the paper in person at our Annual Convention.

Another prize to be awarded by the Research Committee is the annual Ames Award, sponsored jointly by Ames Co. and the College. This year there will be three prizes given to the best three papers published in our official publication, The American Journal of Gastroenterology, between July 1958 and June 1959.

In addition to these two awards, the Research Committee has at its disposal a limited fund for research in gastroenterology. This has been made up from the annual voluntary contributions of the membership of the American College of Gastroenterology. Applications for moneys should be addressed to the Research Committee.

Last, but not least, the College annually awards ribbons and certificates for the best scientific exhibits at our Annual Convention.

All of these are intended to increase and stimulate interest in the field of gastroenterology. Further details concerning these awards will appear from time to time in the Journal or you may obtain them by writing to the Research Committee.

Why not take advantage of the opportunities offered by the American College of Gastroenterology in this field?

Frank J. Borrelli

ABSTRACTS FOR GASTROENTEROLOGISTS

GASTROINTESTINAL TRACT

A 56-YEAR PROGRESS REPORT OF A CASE OF GASTROSTOMY: K. R. Deibert and Lorin Gary. J. M. A. Alabama 28:8 (July), 1958.

The authors present the case of a 17-year old white male who suffered a severe attack of typhoid fever in 1898. This led to the development of marked dysphagia. A gastrostomy was performed in February, 1902 and two later attempts were made to close the stomatal site following treatment of the esophageal stricture by use of bougies and whalebone fillets but they proved unsuccessful. The patient was seen 56 years later and an upper gastrointestinal series in addition to revealing prominence of the phrenic ampulla associated with a hiatus hernia and some esophagitis also showed passage of barium through an outpouching in the fundus to appear on the ventral skin surface. Patient had been hospitalized for the unrelated complaint of pulmonary edema and left ventricular failure.

JOHN M. MCMAHON

SURGICAL TREATMENT OF ORAL CARCINOMA: David E. Sullivan. Northwest Med. 57:901 (July), 1958.

Surgical excision of malignancies about the mouth and jaw, in more recent years, shows an upward trend, as against radiation therapy. Increased interest in surgical treatment is due to improvement of anesthesia, blood replacement, improved technics, and better reconstruction results and antibiotics. There are many places in the country, which are not taking advantage of the recent surgical advances, in treatment of oral carcinomas.

It is impossible to sterilize a carcinoma with radiation once it has invaded bone, without causing osteoradio nécrosis. Also feared is the complication of radiation therapy; for example radiation myelitis, of the cervical spinal cord, following intensive therapy of malignancies about the head and neck. The author believes the only indications of therapy are these: a. Those who refuse surgery or whose condition does not permit surgery. b. Lesions in inacces-

sible areas with palliation.

One of the most encouraging aspects of treatment of oral cancer is in the field of reconstruction. Contractures, distortion of tissues and fistulas can be prevented.

V. J. GALANTE

STOMACH

GASTRIC ULCER: Richard F. Jones, Randolph Clements and Clarence C. Pearson. A.M.A. Arch. Int. Med. 101:855 (May), 1958.

Proponents of surgical treatment of gastric ulcer justify their attitude on the basis of difficulties of differential diagnosis between benign and malignant lesions, the much higher cure rate of small ulcer-like cancers, the low surgical mortality of gastric resection, and the small number of postgastrectomy complications. Therefore, 161 cases of gastric ulcer cases were reviewed some time after termination of either medical or surgical therapy. Fifty-five per cent had surgical, the others medical treatment. The medical cases had been carefully selected by excluding persons with suspicious clinical, laboratory or x-ray findings, recurrent gastric ulcers and ulcer complications. Mortality of the 88 surgical cases was 1.2 per cent; carcinoma was found in 9 per cent of these cases while 4 per cent of the medically treated patients developed carcinoma following their treatment. The conclusion is reached that careful selection of cases, rigid follow-up and proper information given to the patient in regard to medical and surgical risks make it possible to avoid resection in a considerable number of cases.

H. B. EISENSTADT

THE GASTRIC AND JEJUNAL MUCOSAE IN HEALTHY PATIENTS WITH PARTIAL GASTRECTOMY: Fred Lees and L. C. Grandjean. A.M.A. Arch. Int. Med. 101:943 (May), 1958.

Thirty-three persons in good health, having previously undergone a partial gastric resection for ulcer, received a gastric biopsy by the suction tube method. All persons with severe dumping syndrome, anemia, diarrhea, weight loss, anorexia and pain were excluded. Only one of these patients had a perfectly normal gastric mucosa. This was a case of Billroth I resection. The others had mild to severe glandular atrophy and diffuse round cell infiltration. All specimens from the jejunal mucosa were normal. Preoperative mucosal specimens of these and other patients with duodenal ulcers were entirely normal. This suggested that the stump mucosal changes followed the operation. In 4 additional patients with anastomotic ulcer there was no mucosal atrophy of the gastric stump.

H. B. EISENSTADT

X-RAY MANIFESTATIONS OF PEPTIC ULCERATION DURING CORTICOSTEROID THERAPY OF RHEUMATOID ARTHRITIS: Theodore F. Hilbish and Roger L. Black. A.M.A. Arch. Int. Med. 101:932 (May), 1958.

In a series of 68 patients with rheumatoid arthritis 26 per cent had corticosteroid-induced or activated peptic ulcerations. Some ulcers appeared as early as 3 weeks, others as late as 6 years after the beginning of therapy. Typical history and symptoms of ulcer are frequently lacking, only an atypical epigastric pain is frequently but not always present. Occult blood in the stool, unexplained anemia, frank hemorrhage from the bowel, peritoneal irritation, and subdiaphragmatic air accumulation may be the only symptoms of an otherwise silent ulceration. The only objective evidence is frequently a small ulcer crater mostly not tender on pressure during the fluoroscopic examination. Associated x-ray changes usually seen with peptic ulcer such as incisura, increased or decreased peristalsis, edema, radiating mucosal folds, spasticity and deformity are missing. Location of ulcers is not rarely atypical. Microscopically only a sharp punched-out ulcer defect is noticed with minimal surrounding tissue reaction.

H. B. EISENSTADT

GASTROINTESTINAL HEMORRHAGE CASES AT THE SWEDISH HOSPITAL, SEATTLE: Edward B. Speir and Oscar K. Williams. Northwest Med. 57:624 (May), 1958.

The material studied included 303 patients with 191 being male and 112 female. The preponderance of males to females with respect to duodenal ulcer is a ratio of 3 to 1. The males also are afflicted in a greater ratio in gastric ulcer and in gastrointestinal hemorrhage with site unknown in the proportion of 1¾ to 1 and 3 to 1 respectively.

The site of bleeding occurred between the lower esophagus and the ligament of Treitz in 72.5 per cent of all patients with gastrointestinal hemorrhage. Unknown sites account for 16.1 per cent.

The percentage of accuracy of diagnosis with positive x-ray or endoscopy of both was 80.7.

Of the 303 patients in the series, 235 were treated medically and 68 were treated surgically, with an over all operative percentage of 22.4. One hundred ten bleeding duodenal ulcers were treated medically and 25 surgically; 37 gastric ulcers treated medically and 21 surgically; 43 patients whose site of gastrointestinal hemorrhage was unknown were treated medically and 6 treated surgically. Of the remainder 45 were treated medically and 16 surgically.

In the series there were 31 deaths with an over all mortality of 10 per cent. The medical mortality was 8.9 per cent and the surgical mortality was 14.7 per cent. Sixty-two per cent of the medical deaths was due to extenuating hemorrhage, which produced 30 per cent of the surgical deaths.

The site of hemorrhage can be accurately determined in 80 per cent of the patients before laparotomy. It is desirable to stabilize the patient who is bleeding from the gastrointestinal tract and the objective can be reached in 4 out of 5 patients.

JOSEPH E. WALTHER

GASTROPEXIA ANTERIOR GENICULOTA FOR SLIDING HIATUS HERNIA AND FOR CARDIOSPASM: I. Boerema. J. Internat. Coll. Surgeons 29:533 (May), 1958.

The author describes a new operation for this type of sliding hiatal hernia based on the idea that the width of the hiatus is not the important factor in the development of hernias but the important factors are the three forces that bring the stomach up into the mediastinum. The operation that is performed is directed towards counteracting these forces.

The author presents 57 cases undergoing this type of surgery and after re-examination there were only two recurrences. The operation is given in great detail including some very fine drawings showing the various steps in the operative procedure. This particular type of surgery does not require thoractotomy and patients do well during the operative procedure.

ABRAHAM BERNSTEIN

ABDOMINAL SURGERY: Claude E. Welch. New England J. Med. 258:885 (1 May), 1958.

Reports on abdominal surgery are published frequently and the author reviews many procedures. Methods of treating hiatus hernia are discussed, and a series of 113 cases from the Mayo Clinic as presented by Myre, and his co-workers are reviewed.

Evaluation of radical gastrectomy combined with splenectomy and partial pancreatectomy as performed by McNeer and his co-workers are presented. Mortality rate and lymphatic spread in 7 cases is reviewed. Nakayama in Japan records a low mortality rate in 329 gastrectomies.

Discussion of the value of exploratory gastrotomy is found pertinent and operative procedures in gastric and duodenal ulcers are described.

Temporary gastrostomy by means of a Foley-catheter is substituted for the post-operative Levin tube decompression and 115 such cases are reported by Farris and Smith.

The literature is reviewed and statistics presented on strangulation obstruction, ileocolitis, ileoentectropy, carcinoid tumors, ruptured appendices, ileocecal lipomatosis, ulcerative colitis, perforation of the cecum and subtotal colectomy.

ABRAHAM BERNSTEIN

ABDOMINAL SURGERY (CONCLUDED): Claude E. Welch. New England J. Med. 258:939 (8 May), 1958.

The author concludes his review on abdominal surgery in this article. He covers the literature and reviews the statistical study in portal hypertension with emphasis on bleeding varices and methods of therapy. Liver surgery is described in detail.

Brief references are made to surgery of the biliary tract.

Papers on splenic rupture, injuries to the liver, gallbladder and pancreas are reviewed.

The importance of adrenal insufficiency in surgical patients is emphasized. Articles on peritonitis and mesenteric-artery infarction are reported. The author has done an excellent job in reviewing these various papers on abdominal surgery and gives the reader a very excellent summary. Complete references are listed and all articles in their entirety can be referred to. Practically all diseases in the domain of abdominal surgery are discussed.

ABRAHAM BERNSTEIN

TREATMENT OF GASTROINTESTINAL DISORDERS: AN EVALUATION OF SUSTAINED-ACTION MEDICATION: Charles W. Hock. Am. Pract. & Digest. Treat. 9:940 (June), 1958.

The author reviews the results in 82 cases. He states that good to excellent results were obtained in 53 or 75.7 per cent of his 82 patients while moderate relief was evidenced in another 12 patients.

In all the cases a careful history was recorded, followed by a complete physical examination, including x-ray and sigmoidoscopic, and laboratory studies.

Management of gastrointestinal disorders, especially functional bowel distress, was done in three ways—1. practical psychotherapy, 2. proper diet, and 3. anticholinergic plus sedative therapy. Antacids constituted a part of the regimen in cases of peptic ulcer.

We would feel that the good results obtained in this report are not entirely due to anticholinergic drugs alone but a combination therapy which has been found useful for a long period of time by previous authors.

I. HENRY EINSEL

INTESTINES

PERFORATION OF THE GASTROINTESTINAL TRACT BY AN UNUSUAL FOREIGN BODY—A PORCUPINE QUILL: Harry C. McDade and Walter B. Crandell. New England J. Med. 258:746 (10 Apr.), 1958.

The authors report two cases of perforation, one occurring in the distal segment of the jejunum, the other in the stomach, both of which were primarily produced by a porcupine quill.

In both cases the patients had eaten a sandwich, one a porcupine meat sandwich, the other a sandwich following the handling of the porcupine, in which a fragment of a quill was swallowed with the sandwich.

The frequency of ingestion of porcupine quills is unknown, but must be very rare. The authors were able to find only two other case reports of this condition.

The precise preoperative diagnosis is rarely made. In both of the authors' cases the information that the patient had swallowed a porcupine quill was made after postoperative inquiry.

ZACH R. MORGAN

ROENTGEN DIAGNOSIS OF ILEAL INTUSSUSCEPTION: L. G. Rigler and H. W. Godfrey. Am. J. Roentgenol. 79:837-841 (May), 1958.

The mortality rate in intestinal intussusception for the first 24 hours is only 7.7 per cent. After 48 hours, the mortality rate is 54 per cent. Ileal intussusception is

uncommon and it occurs in about 9 per cent of gross series of 702 cases.

In at least 80 per cent or more of all cases, the methods of roentgenological examination necessary for diagnosis are those of plain film of the abdomen, barium enema and barium meal examinations.

In the plain film of the abdomen the specific roentgen sign is an area of increased radiability having the shape of a beak resulting from a conical tapered narrowed proximal lumen of the intussusception distended by gas. Sometime this shadow may be linear or curvilinear within a soft tissue mass rather than conical or funnel-like narrowing.

Barium meal examination with a follow-up examination of the small bowel shows a delay of the barium column which narrows as it enters the proximal intussusceptum is an important finding. Other times the barium tends to flow in a retrograde fashion between the walls of the intussusceptum and the intussuscipiens which results in coilspring outline of the intussusceptum mass.

The roentgen finding obtained by barium enema depends entirely on reflux through the ileocecal valve into the terminal ileum. The resultant picture is that of a coilspring appearance.

VINCENT J. GALANTE

ARTERIAL MESENTERIC OCCLUSION OF THE DUODENUM: W. P. Bitner. Am. J. Roentgenol. 79:807-813 (May), 1958.

This condition results in intermittent occlusion of the duodenal loop with a compression of the third portion of the duodenum by the superior mesenteric artery. At the point where it crosses over the loop with the superior mesenteric vein and nerve, all show clinical improvement following surgery.

Symptomatology is variable, from gaseous indigestion to pain. In the roentgenological examination, the following criteria are important in the diagnosis:

1. Constant dilatation of the duodenum proximal to the mid part of the transverse portion of the duodenum in which there is contact with the superior mesenteric artery sheet.
2. Normal duodenum distal to this site.
3. Retention of the barium beyond the normal time limit.
4. To and fro movement of barium within the duodenal loop.

VINCENT J. GALANTE

ACUTE AND CHRONIC THROMBOSIS OF THE MESENTERIC ARTERIES ASSOCIATED WITH MALABSORPTION: R. S. Shaw and E. P. Maynard, III. New England J. Med. 258:874 (1 May), 1958.

Thromboendarterectomy of a major aortic branch is an established surgical procedure and even preferred to segmental resection if there is any degree of patency. This experience has been chiefly with iliac and femoral arteries as well as the aorta.

The authors report successful thromboendarterectomy in two cases involving the superior mesenteric artery, one acute and one chronic thrombotic lesions.

Visceral thrombotic lesions should be considered in patients with unexplained malabsorptive states inasmuch as thromboendarterectomy may represent an effective therapy in these patients, as well as those in whom mesenteric vascular thrombosis results in intestinal infarction.

A. M. SUSINNO

ACUTE VOLVULUS OF THE CECUM: James H. Saint. Am. J. Surg. 95:798 (May), 1958.

A case is reported of acute volvulus of the cecum in a 59-year old white woman. The diagnosis was made preoperatively. The writer stresses the important clinical features of acute volvulus of the cecum which is a relatively uncommon condition. The mechanism is described in detail. The outstanding general characteristic of the symptoms of acute obstruction produced by an acute volvulus of the cecum are: sudden onset of sharp nauseating type of pain, with rapid increasing severity, as a

result of the distention of a thin wall viscus. This gives rise to a striking abdominal asymmetry, that is, a circumscribed swelling in the region of the right lower abdomen. This is unlike any other type of intestinal obstruction.

Evidence is also given that a flat plate of the abdomen is of considerable help in diagnosis, and that a barium enema is not absolutely necessary, but may be confirmatory.

CARL J. DEPRIZIO

CLINICAL CONSIDERATIONS OF ABDOMINAL PAIN: James M. Northington. Clin. Med. 5:473 (Apr.), 1958.

The author feels that any pain in the epigastric region lasting more than two weeks should cause suspicion of cancer of the stomach, especially if it occurs in a middle-aged man. This lesion rarely gives severe pain until late in the course of the disease.

Cancer pain is usually persistent and intensified by eating, but sometimes will mimic ulcer pain with relief temporarily after the intake of food.

In the consideration of abdominal pain, one can not ignore the small intestine, but when pain is due to small intestine disease, it is rarely because of tumor. In a series of 300 patients with large bowel cancer, 57 per cent complained of abdominal cramping. The distribution of cancer in these patients was about equal in the right and the left half of the colon so it becomes imperative that we look high in the colon for evidence of cancer. The author goes on to state that in patients over 40 years old with no previous history of abdominal surgery, the most common cause of intestinal obstruction is cancer of the colon. In two-thirds of these, there will be an insidious, gradually increasing change in bowel habits usually presenting the picture of alternate constipation and diarrhea.

In any man over 50, symptoms suggestive of appendicitis should call to mind the possibility of a cancer of the cecum or appendix.

Mid-epigastric pain radiating to the back and relieved only by narcotics or severe flexion of the thighs against the abdomen may be indicative of pancreatic cancer. In this case, the cancer in the head of the pancreas will likely radiate into the right upper quadrant but unfortunately jaundice may long precede the occurrence of pain in these patients. Cancer in the tail of the pancreas has a more gradual onset and when pain occurs it almost always radiates into the left hypochondrium and to the left side of the chest. Primary cancer in the liver seldom produces pain while in about one-third of the adenocarcinomas of the kidney, pain is the first symptom. In this instance, it is also rare to see the patient who does not already have hematuria and a palpable tumor mass.

Acute pain in the abdomen of women is usually not due to cancer. When it is present, the growth is usually a myoma of the uterus or an adnexal tumor. Pain is not an early symptom in cases of cancer of the endometrium or cervix.

L. K. BEASLEY

CLINICAL AND SURGICAL VIEWS ON ASCARIASIS, A CONTINUING PROBLEM: David Wyatt Aiken. Clin. Med. 5:515 (Apr.), 1958.

The author points out the necessity for doctors being alert to the possibility of ascariasis all over the United States inasmuch as there have been large migrations of population of the lower income group from the South and from the West Indies. He goes on to point out that partial obstruction of the small intestine is the commonest complication of ascariasis and usually responds very well to treatment with piperazine compounds which narcotize the parasites and allow their disentanglement from a bolus which might be too large to pass on. He prefers this drug in preference to hexylresorcinol which will kill the ascarids promptly leaving a bolus present which may have to be removed surgically.

His diagnostic examination consists of two procedures, namely, examination of the stool for ascarids, and x-ray examination of the small intestine one or two hours after the administration of four drams of barium mixture by mouth.

L. K. BEASLEY

A NEW FORMULA FOR PRURITUS ANI ET VULVAE: Laurence L. Palitz. Clin. Med. 5:635 (May), 1958.

An ointment (hydrocortisone, estrone, Vitamin A and pyrilamine maleate) was prescribed for use on pruritus areas including pruritus ani et vulvae, etc.

The preparation was effective immediately in a high percentage of the cases and the relief obtained maintained for a considerable time.

IRVIN DEUTSCH

INTUSSUSCEPTION IN ADULTS: Harold P. McGinnes. Illinois M. J. 113:221 (May), 1958.

An excellent review article covering the subject of intussusception including a classification of the various types and their frequency of occurrence, the possible etiology, the signs and symptoms and technic of management. The article also includes two interesting case presentations. The article acts as a stimulus to consider possible intussusception in the differential diagnosis of adults experiencing intermittent crampy abdominal pain without a demonstrable primary lesion.

RALPH D. EICHHORN

HEMOPERITONEUM: A DIFFERENTIAL DIAGNOSIS WITH NOTES ON SURGICAL MANAGEMENT: Lawrence G. Khedroo. Illinois M. J. 113:225 (May), 1958.

The author presents a review article of hemoperitoneum. He lists the causes as ectopic pregnancy, ruptured Graafian follicle, hemorrhagic acute pancreatitis, metastatic peritoneal carcinomatosis, superior mesenteric vessel thrombosis, acute thrombocytopenic purpura, volvulus, endometriosis and trauma. He discusses the differential diagnosis of each and the points which will guide a surgeon as to the etiology of the hemoperitoneum. This is important since the preoperative consideration of these factors can decrease the operating time and prevent additional peritoneal irritation associated with prolonged exploratory maneuvers. Knowledge of the mechanisms involved provide a rapid assessment and a rational approach in hemoperitoneum.

RALPH D. EICHHORN

INTRAABDOMINAL HEMORRHAGE FOLLOWING A CONTUSION OF THE ABDOMINAL WALL: T. K. Whaley and O. C. Colt. Central African J. Med. 4:200 (May), 1958.

It is known that contusion of the abdominal wall may result in injury to the underlying viscera. The contusion may be due to a direct blow or the shock wave of air or underwater blast. This interesting report features a case where a large amount of fresh blood, in addition to clotted blood within the peritoneal cavity at the time of the operation, no active bleeding point was located. We must therefore conclude that the hemorrhage was the cumulative effect of bleeding from the grossly lacerated abdominal wall, the torn omentum and the retroperitoneal hemorrhage.

From a diagnostic point of view, it may be noted that there was an interval of approximately 24 hours between the original injury and the onset of unequivocable signs and symptoms of intraabdominal damage.

JOHN E. COX

NATURAL HISTORY OF DIVERTICULOSIS OF THE COLON: John L. Horner. Am. J. Digest. Dis. 3:343-350 (May), 1958.

The paper is a timely and valuable aid out of the confusion that seems to exist with regard to the potential seriousness of this anatomic disorder. The author's conclusions deserve particular attention as they are the result of observations and follow-up studies of up to 18 years on 503 patients seen in private practice. Two-thirds of his patients showed no significant increase in number, size, or distribution of

the diverticula over up to 14 years of observation. Attacks of classical diverticulitis occurred in 85 patients or 17 per cent. Of these only two patients, 0.4 per cent of the total, required surgery. There were 53 per cent females against 47 per cent males. The high incidence of hiatus hernia among his patients makes the author feel that increased intraabdominal pressure is at least a contributing factor in the development of diverticulosis coli.

WALTER CANE

LIVER AND BILIARY TRACT

ACUTE CHOLECYSTITIS: Meredith J. Evans. Med. Times 86:313 (Mar.), 1958.

In contrast to the viewpoint held previously, surgery is now advised in the first 48 to 72 hours after onset. The rationale for early operation is that invasive bacterial infection appears at this time, and that, in spite of the inflammatory process, it is easier and safer to dissect the gallbladder at this stage than when it is distorted by dense adhesions.

In preparing the patient for surgery, nasogastric suction should be instituted and continued postoperatively; anemia and fluid and electrolyte imbalances should be corrected by parenteral whole blood and glucose containing the necessary electrolytes; and antibiotics should be used routinely, usually penicillin. A mixture should not be used, especially not one containing streptomycin, since gram negative organisms rapidly develop a resistance to it.

The procedure of choice is cholecystectomy. A cholecystostomy may be performed if the patient's condition is so deteriorated as to contraindicate prolonged anesthesia, or if the operator is unfamiliar with the anatomy and anomalies of the portal triad.

WALTER LENTINO

SOLITARY BENIGN ADENOMA OF THE LIVER ASSOCIATED WITH PROGRESSIVE HEPATIC INSUFFICIENCY: B. V. Jager and C. A. Nugent. A.M.A. Arch. Int. Med. 101:645 (Mar.), 1958.

A 19-year old white girl was suffering from severe general pruritus, in addition she had attacks of low-grade fever, general malaise and jaundice. The liver was considerably enlarged and tender. Liver battery tests and biopsy did not help make a diagnosis. Surgical exploration revealed the benign tumor of the liver, a liver cell adenoma, the excision of which was attempted but could not be completed because of its large size as well as of the severe bleeding. Patient died a few hours after the operation.

Benign adenoma of the liver occurs most frequently in children and young adults. It rarely grows to a size interfering with liver function. It may be difficult to distinguish liver cell hyperplasia, benign tumor, and malignant hepatoma from the liver biopsy.

H. B. EISENSTADT

PATHOLOGIC ASPECTS OF CIRRHOSIS: Hans Popper and Frederick G. Zak. Am. J. Med. 24:593 (Apr.), 1958.

Cirrhosis is characterized by interdependent parenchymal and mesenchymal processes. Fibrosis in cirrhosis may be the result of primary irritation of the connective tissue or of increased pressure in the hepatic vein tributaries, or it may be stimulated by epithelial alterations such as hepatocellular necrosis with subsequent stromal collapse, degeneration or fatty metamorphosis of liver cells, and excess of ductular cells. Anatomically, cirrhosis is defined by an altered reconstruction of the tubular architecture reflected in degenerative nodules and in connective tissue septums linking portal canals and centrolobular fields and carrying vascular anastomoses. The formation of nodules and septums is dovetailed. Either may be active or passive in relation to the other.

Cirrhosis is best classified according to: 1. Histogenesis, 2. Etiologic factors and 3. Functional alterations. The histogenetic classification depends upon the three pathways through which the architecture is re-

constructed (postnecrotic, diffuse septal and biliary). The etiological classification is hampered by insufficient knowledge of the etiological hepatic failure (either hepatocellular or hepatocirculatory), portal hypertension, cholestasis, extent and activity of the cirrhotic process. In respect to prognosis and therapy the extent of cirrhosis is of less significance than the activity of the cirrhotic lesion. This is an excellent review for those interested in the problem.

JOHN M. MCMAHON

VIRUS HEPATITIS: Lucian M Ferris. Clin. Med. 5:319 (Mar.), 1958.

A brief article reports on 109 cases of virus hepatitis occurring in Vicksburg, Mississippi in the past eight years, of which eight were cases of homologous serum hepatitis, the others, infectious hepatitis.

The stages of the disease are discussed briefly.

Very little is written concerning differential diagnosis or laboratory findings except for the thymol turbidity and cephalin-cholesterol flocculation.

Nothing new is added in therapy and while the vast majority of these patients recover, there are still a few cases that are fatal.

PAUL LEDBETTER

INTRAVENOUS CHOLANGIOGRAPHY IN DETECTION OF STONE-BEARING CYSTIC-DUCT REMNANTS (SO-CALLED REFORMED GALLBLADDERS): J. Edward Berk and Robert N. Lee. Am. J. Digest. Dis. 3:220-228 (Mar.), 1958.

Dilated cystic-duct remnants or so-called reformed gallbladders containing calculi may be demonstrated by means of intravenous cholangiography in patients who have undergone cholecystectomy. Stone-bearing dilated cystic-duct remnants are one reason why postcholecystectomy syndrome cases occur. The authors emphasize the need to explore the biliary ducts diligently for calculi at primary cholecystectomy and to remove the entire gallbladder and as much of the cystic duct as possible.

WALTER CANE

THORAZINE JAUNDICE: Kirk V. Cammack, John W. Hoffman and Max Dodds. J. Michigan M. Soc. 57:582 (Apr.), 1958.

Chlorpromazine can produce a jaundice indistinguishable from obstructive jaundice, by commonly used laboratory tests. The author cites 2 cases, which were operated on with disastrous results.

Thorazine jaundice develops independently of dosage and usually appears 2-3 weeks after administration of the drug and is usually preceded 4-5 days by chills and fever, anorexia, nausea and vomiting, pruritus and malaise. There may be a history of allergy and a peripheral eosinophilia. Liver biopsy shows cholestasis with infiltration of leucocytes and slight or no degeneration of liver cells or fibrosis.

SAUL A. SCHWARTZ

STUDIES ON INFLUENCE OF PROTEIN ON THE LIVER INJURY DUE TO CHLOROFORM AND CARBON TETRACHLORIDE: Takashi Nakamura, Shozo Nakamura and Etsuko Kawamura. Tohoku J. Exper. Med. 67:373 (25 Apr.), 1958.

In a series of experiments by the authors, attempts were made to determine the influence not only of high and low protein diets in experimental animals later subjected to the liver poisons chloroform and carbon tetrachloride but also to find out if there is any effect that could be correlated with the length of time the animals were fed these experimental diets. Rats were fed 5 per cent casein and 30 per cent casein diets for five days, two weeks, 4 weeks and 8 weeks, and then given chloroform or carbon tetrachloride by stomach tube. Rats who died were autopsied and any survivors were killed after three days in order to study the liver and kidney pathology in those rats receiving chloroform. The instance of liver necrosis in a group which were fed 5 per cent casein diet for four weeks or eight weeks prior to chloroform was significantly higher than those rats which were fed 30 per cent casein. With

carbon tetrachloride, however, the reverse was true. They found that kidney injury was generally lighter than liver injury and that it was milder in carbon tetrachloride poisoning than in chloroform poisoning. The instance of necrosis of the epithelium of the renal tubules was significantly lower in those rats fed with 5 per cent casein for two weeks. Death rate after the administration of the poisons failed to run parallel with the degree of liver injury and was also not proportional to the degree of kidney pathology. The authors feel that although chloroform and carbon tetrachloride are closely related chemically, there is considerable difference in their mode of action as parenchymal poison.

PAUL B. VAN DYKE

ACUTE FATTY METAMORPHOSIS OF THE LIVER ASSOCIATED WITH PREGNANCY: (CPC) Northwest Med. 57:456 (Apr.), 1958.

This is the 15th case reported of this unusual condition designated as "acute fatty metamorphosis of the liver associated with pregnancy". This patient was an 18-year old female who delivered a dead child. The story was complicated by a traumatic delivery, sedation, transfusions, jaundice, coma and exanguination hemorrhages. The bleeding was never controlled, many transfusions were given; hysterectomy resorted to, and finally liver failure and death.

The case report is well discussed by the physicians present. At post, the liver was shrunk; there was no necrosis of the liver cells, but liver cells were distended with vacuoles indicating "fatty infiltration". As to the cause; this is uncertain. It has occurred in tetrachloride intoxication, protein-deficient diet, high ethionine diet. All these were on experimental animals with production of fatty degeneration of liver. This "fatty metamorphosis of liver" associated with pregnancy is a new entity—possibly due to alteration in endogenous metabolism. The postulated cause of liver failure—methionine, cholene and vitamins—keep liver going—at term tremendous amount of estrogen has to be detoxified, this puts load on the liver causing fatty metamorphosis and liver failure.

LIONEL MARKS

PERITONEOSCOPY IN THE STUDY OF LIVER DISEASE: Eric R. Sanderson. Northwest Med. 57:611 (May), 1958.

Problems in diagnoses can be resolved by either needle biopsy, laparotomy or peritoneoscopy. Of these methods, peritoneoscopy is superior with respect to relative morbidity and knowledge obtained. It is possible to observe both lobes of the liver, examine the gallbladder, take specimens of the liver in diseased areas rather than blindly and inject radiopaque material into the gallbladder to visualize the biliary tree. It is also possible to make splenoportograms in order to determine the presence of varices and the site of obstruction in the portal venous system.

JOSEPH E. WALTHER

BILE PERITONITIS: Raymond E. Anderson. Northwest Med. 57:595 (May), 1958.

Extravasation of bile into the peritoneal cavity results in bile peritonitis. The entity constitutes a very serious clinical problem which results in a mortality rate of 50 to 75 per cent if untreated. Hall is quoted as saying that 90 per cent of patients will recover if operated upon within the first 24 hours after onset; survival rate is 60 per cent if 24 hours elapse and 24 per cent if delay is continued for 36 hours. The etiology is fully discussed.

Clinical symptoms may be divided into acute, subacute and chronic periods. The acute period is characterized by sudden agonizing abdominal pain, nausea, vomiting and shock. Abdominal distention, tenderness, evidence of spreading peritoneal irritation and ileus rapidly follow. The temperature may elevate alarmingly. In the subacute period the abdomen becomes less tender with a dull aching distress replacing the agonizing pain. A doughy consistency to the abdomen replaces the tense distention of the initial acute stage and shifting dullness may be elicited. Hiccups, jaundice, extreme lethargy and mental deterioration

often occur. In event of survival, the chronic period occurs characterized by constant exudation of peritoneal fluid with resulting ascites, chronic adhesive peritonitis and external biliary fistulas.

Successful therapy demands immediate surgical drainage.

JOSEPH E. WALTHER

PSYCHOSOMATIC MEDICINE

PATHOGENESIS IN HUMAN OBESITY: Albert J. Stunkard and Harold G. Wolff. Psychosom. Med. 20:17 (Jan.-Feb.), 1958.

This report deals with a mechanism of satiety and how it may become disordered in human obesity. It studies the theory that a major factor regulating food intake is the amount of carbohydrate available for use by the individual. In this study an effort was made to determine the effects of intravenous administration of glucose on gastric hunger contractions in human subjects. Correlation of the capillary-venous glucose differences with the effects of glucose administration on gastric hunger contraction provides evidence that satiety may be a function of the metabolic events accompanying the increased utilization of glucose. Secondly, intravenously administered glucagon in every one of thirty consecutive patients abolished gastric hunger contractions in the experience of hunger within three minutes.

The authors conclude that their work tends to confirm the widely held impression that obesity represents an inappropriate adaptive response and presents evidence that in some persons this response involves a disturbance in carbohydrate metabolism.

RALPH D. EICHHORN

BOOK REVIEWS FOR GASTROENTEROLOGISTS

DIETETIQUE THERAPEUTIQUE: J. Tremolieres, A. Mosse, L. Debbes with the collaboration of J. Claudian, C. Flament, J. Paschoud, G. Pequignot, F. Vinit—Preface by Professeur E. Le Breton. 555 pages, paper covers. G. Doin & Cie., Paris, France, 1958.

This book is one of a series dealing with the treatment of the various disorders of the system including preoperative and postoperative conditions by means of diet. There are numerous tables and formulas which may be useful to physicians who read French.

The book is well printed and has an adequate bibliography including American literature.

CLINICAL ENZYMOLOGY: Edited by Gustave J. Martin, Sc.D., Research Director, The National Drug Company, Philadelphia, Pa. 241 pages, illustrated. Little, Brown & Company, Boston, Mass., 1958. Price $6.00.

Clinicians and biochemists will find useful material in Martin's Clinical Enzymology.

Dr. Martin, in the last paragraph of the introduction, sums up the basic knowledge essential to an understanding of the field of enzymes.

The reviewer found the book interesting and instructive and recommends it as a useful reference text to the physician's library.

DIAPHRAGMATIC HERNIAS IN ADULTS AND CHILDREN: Alfredo Cesanelli, M.D. and Juan Jose Boretti, M.D. 344 pages, illustrated. Actas de La Association Argentina de Chirugia, 1958.

Although written in Spanish, with the aid of Spanish-English dictionary, the reviewer was able to decipher most of the text. It is one of the best presented books dealing with herniations of the diaphragm and it is recommended that an English edition be brought out by the authors.

Clinicians and surgeons will find many new and useful suggestions within these pages.

TRANSACTIONS OF THE 6TH MEETING OF THE INTERNATIONAL SOCIETY OF GEOGRAPHICAL PATHOLOGY, PARIS 9-11 JULY 1957: 642 pages, illustrated. S. Karger AG, Basel and New York, 1958.

A symposium on ulcer and various other aspects of the stomach by well known clinicians given under the auspices of the International Society of Geographical Pathology in Paris, France.

This symposium should be of great interest for all physicians, whether or not they specialize in gastroenterology, roentgenology or biochemical research.

DIAGNOSTIC GASTROENTEROLOGY: Fernando Milanes Alvarez, M.D., Auxiliary Professor and Chief of Clinic of the Hospital Calixto, Garcia, Havana, Cuba. 940 pages, illustrated. Editorial Universitaria, University of Havana, 1958.

Dr. Milanes, who is a well known gastroenterologist and a Governor of the American College of Gastroenterology, has written an excellent book dealing with gastrointestinal diagnosis and treatment. Physical examination and various tests of the gastrointestinal tract, diseases of the liver, pancreas and parasitology, diagnosis and treatment, are well presented.

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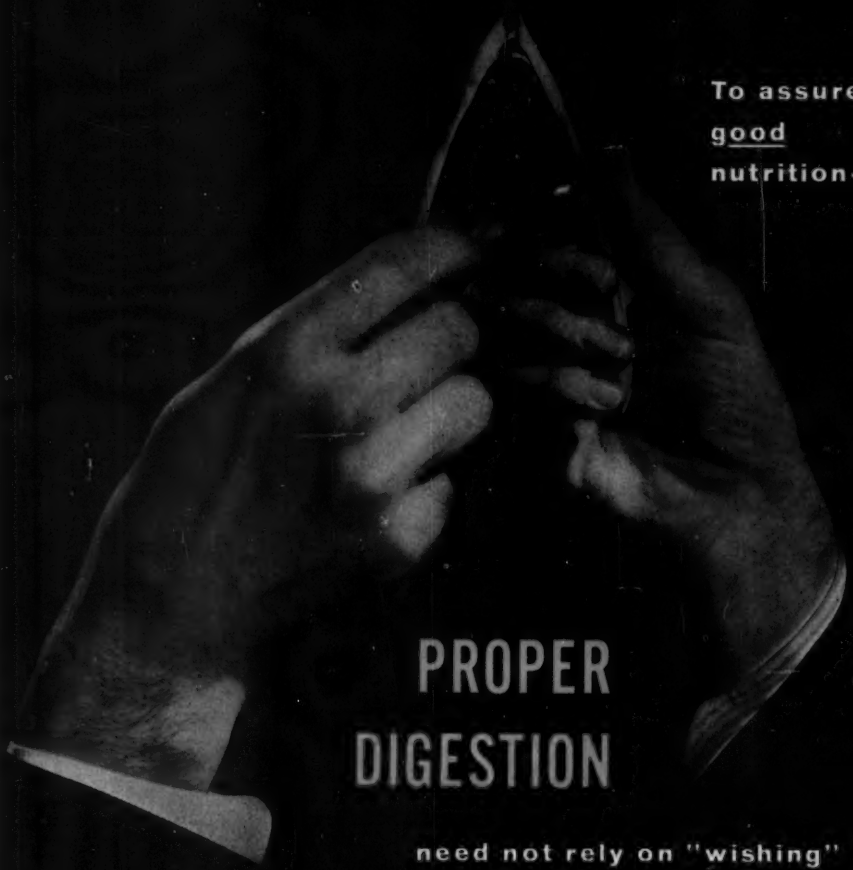

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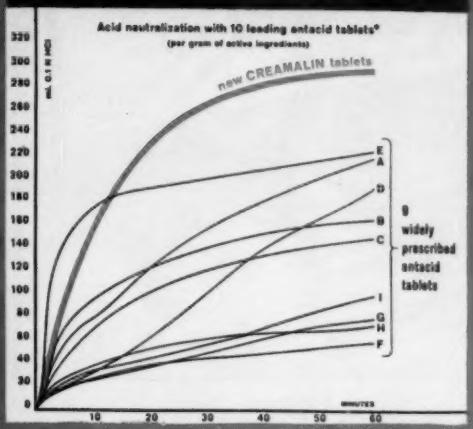

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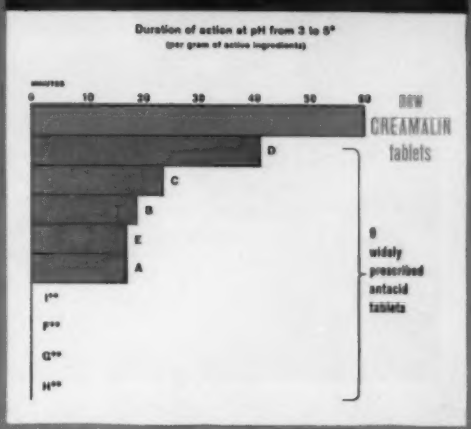

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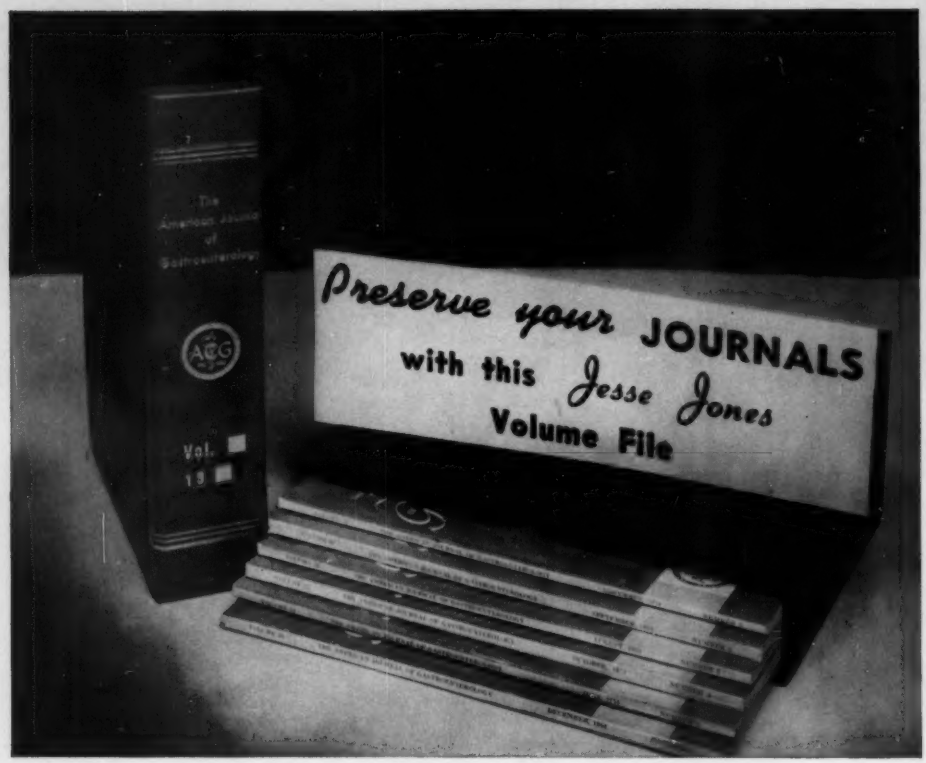

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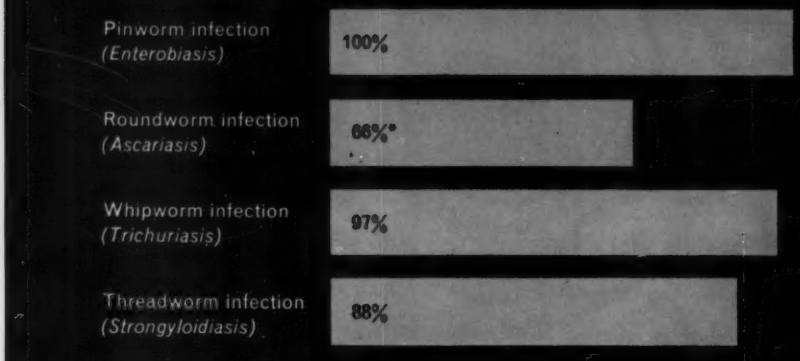

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References: 1. Crunk, G. A.; Naumann, D. E., and Casson, K.: Antibiotics Annual 1957-1958, New York, Medical Encyclopedia Inc. 1958, p. 397. 2. Newcomer, V. D.; Wright, E. Y., and Sternberg, T. H.: Antibiotics Annual 1954-1955, New York, Medical Encyclopedia Inc., 1955, p. 686.

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